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THE VERTEBRAL VEIN SYSTEM*

CALDWELL LECTURE, 1956

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Eugene Wilson Caldwell

In the summer of 1919, I came upon the book by Pusey and Caldwell[52] on Radiology. At that time, double-coated roentgenographic films were still a novelty and the use of two intensifying screens was uncommon. To my surprise, in this 1904 edition, Dr. Caldwell, then Mr. Caldwell, described in detail the use of double-coated films and two intensifying screens. I learned also of the Caldwell stereofluoroscope. Later that same summer I read of the improvements in this stereoscope[44] in the 1918 volume of the AMERICAN JOURNAL OF ROENTGENOLOGY. These improvements are still in use. This volume also contained the obituary notices[3,56,57] of Dr. Eugene Wilson Caldwell, former President of the American Roentgen Ray Society. In 1918 the roentgen ray was twenty-three years old. Dr. Caldwell had been a practicing radiologist for twenty years. In his forty-eight years, he made notable contributions in the field of electrical engineering and in the technical and the medical sides of radiology. It is fitting that his memory and his accomplishments be recognized in a lectureship. I feel greatly honored to have been asked to give this lecture.

LIVING anatomy is slowly editing and replacing the anatomy of the dead room. Roentgenology is increasingly responsible for this. Much of gross physiology was founded upon shrewd guesses as to what might be. Some of these guesses have been confirmed, yet many of our most cherished notions about function are the result of someone's effort to prove that the reactions of the organism are purposeful and are designed to achieve a supposed beneficial result. It is but recently that Drabkin[24] had the temerity to state, " . . . the body is not always wise and the environment often unfit."

The concept of a vertebral vein system has to do with this living anatomy. An anatomic system is a complex of structures that has certain functions in common or structures which in their totality contribute to a recognized function. At first, the justification of the concept of a vertebral vein system by experiment while definite was

* From the Department of Anatomy in the Graduate School of Medicine at the University of Pennsylvania. Presented at the Fifty-seventh Annual Meeting of the American Roentgen Ray Society, Los Angeles, California, September 25–28, 1956.

Fig. 1. Vesalius was the first to illustrate veins leaving the spinal canal. Vidus-Vidius first illustrated the longitudinal sinus. He showed one cross connection. Willis showed the cross connections for the entire canal.

limited. Today, sixteen years later, the concept is adequately confirmed by constantly increasing observation in patients and by experimental observations in animals.

It seems incredible that a great functional complex of veins would escape recognition as a system until 1940. On closer appraisal, one explanation appears. The vertebral veins* are the unifying component of the vertebral vein system. They have either been unknown to, or ignored by, the majority of anatomists because special methods must be used to display them. Parenthetically, by roentgenography, the anatomy of the vertebral veins is displayed readily.

When I first presented the concept of a vertebral vein system, I credited Gilbert Breschet[14,15] as being "the first to fully appreciate the complexity and interrelationship of these veins of the skull and vertebral column." The early history of these vessels can best be stated by quoting Breschet[15] himself, in translation.

History of the Vertebral Veins

"Sylvius[58] and Vesalius[60] appear to be the first to have observed the branches which the vertebral veins send into the spinal canal. After them, *Falloppio*[27] found the longitudinal vertebral sinuses (sinus vertebrales longitudinales) in the cervical region. Much later, Vidus-Vidius[61] discovered a transverse vertebral sinus on the third cervical vertebra. Above all, however, the honor goes to Willis[66,67] for having described and represented, although most imperfectly, the longitudinal and transverse sinuses for the entire length of the spinal cavity. Next, after this English anatomist, we are in debt to our compatriot Vieussens,[62] Professor in the Faculty of Medicine at Montpellier, for a description less inexact and more precise of these same parts of the spinal venous system. Since then, these venous channels have fallen into oblivion. They have not been mentioned in the elementary works of anatomy nor demonstrated in oral lessons. We are indebted to M. le Professor Chaussier[17] for having called anew the attention of anatomists to these important vessels, and to him

* The term "vertebral veins" is used here to mean the internal and external complex of veins of the spinal column (described later) and to include the small "vertebral veins" of the transverse foramina of the cervical vertebrae.

for the description of their distribution and their function.

"One sees with amazement that in the majority of our better manuals of Anatomy, nothing is said. Thus Winslow, Sabatier, the MM. Boyer, Portal, Soemmering, John and Charles Bell, Alexander Monro, and so on, have not made any mention in their works of the plexus and sinuses of the spinal veins, nor the venous canals of bone.

"Buisson,[16] to whom we owe the editing of the fourth volume of the "Descriptive Anatomy" of Bichat, gives a sufficiently lengthy account of the great anterior longitudinal spinal veins, but an account inexact and incomplete in many points. Neither J.-F. Meckel[43] nor H. Cloquet[19] have made scarcely any change in this description, although J. Christian Loder,[40] in 1803, has depicted in his Atlas of Anatomy, figures which approach the truth better than those of other anatomists.

"It is surprising that so able an anatomist as Bock retained in the work which he recently produced, the figures published by Loder. In the drawings and in the descriptions of the spinal venous system, we do not find the exactness and precision of the historian of the fifth pair of nerves.*

"In effect, in Plate VII, Bock has attempted to show the great spinal veins or venous sinuses of the spine, but these figures have not been prepared from actual dissections. As with all anatomists who preceded him, he has considered the basi-vertebral veins to be a simple anastomotic branch between the two great longitudinal venous sinuses. These basilar veins, however, have their distribution in the bony substance of the bodies of the vertebrae and communicate with the veins located on the anterior or pre-spinal surface of the spine. The error of Bock is difficult to understand, since before the publication of his work, I recorded a description of the intra-rachidian veins in a thesis delivered in formal competition."†

FIG. 2. Vieussens' figure and description were less "inexact." Breschet gave an excellent description and the best illustrations of the vertebral vein system up to that time in 1819. Bock, a compiler, in 1823 still showed the cross connection as a simple tube.

No one who has seen the superb anatomic plates of Breschet would have any hesitancy in according him first place in establishing the detailed and intimate anatomy

* This sarcasm refers to an earlier publication of Bock.[12]

† A competition to fill the post of Chef de Travaux Anatomiques in Paris. This "Essai sur les veines du rachis" was delivered April 28, 1819, and published[14] the same year.

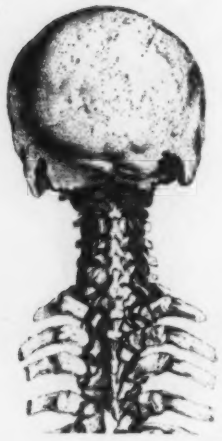

Fig. 3. The venous plexus on the posterior of the vertebral column. Reproduced from part of a one half life size lithograph in Breschet's 1819 article.

of the veins of the spinal canal and the vertebrae themselves.* Breschet's publications are not widely distributed and his major work is incomplete.

In 1818, Breschet was a competitor for the post of Chef de Travaux in the Anatomy Laboratory in Paris. He chose for one of his lectures in the competition, the subject of the vertebral veins. He describes the lack of knowledge of this topic and notes that in earlier competitions, this topic had been assigned twice by the judges. He mentioned Chaussier's synoptic table[17] and Dupuytren's lectures.[26] The published thesis was illustrated by lithographs, one-half natural size. These figures (Fig. 2 and 3.), while good, are much inferior to his later ones. In the thesis, Breschet gives precise details on how to inject the veins of the spine for study.

In 1828, Breschet, now Chef de Travaux, began as a serial publication, a truly monumental work on veins. This publication was to have had twenty-two parts. Unfortunately, only eight parts were completed. I know of six copies in the United States and I have had the opportunity of examining four of these. In the copies that I have seen, the plates have been colored by hand. All the figures are life size (Fig. 4 and 5). The spinal veins are accurately and beautifully shown. I can confirm the essential accuracy of the plates, but I might add that with the new injection materials I now have available, even the great mass of vessels that Breschet illustrated fall far short of the actual number present. Eponymically, the veins in the vault of the skull are known as Breschet's veins. We commonly forget that the veins in the bodies of the vertebrae are likewise Breschet's veins.

Here, in brief review, is the anatomy of the vertebral veins as Breschet left it. The veins are a plexiform network; their numerous components have been adequately named. While the blood in them eventually reaches the heart, the veins do not converge

Fig. 4. The longitudinal venous sinuses with cross- and circle-like connections. EM, emissary connections into vertebral bodies; EX, posterior external plexus. Reproduced from Breschet's life-sized lithograph, 1828. The letters and arrow have been added.

* It is surprising that so able an anatomist as my friend H. A. Harris,[22] (to paraphrase Breschet) would refer to Bock's work as "monumental." Professor Hyrtl said Breschet's book was the only "Hauptwerk" on veins. Perhaps Professor Harris has not seen Breschet's "Essai" nor the beautiful life-size colored lithographs of Breschet's larger work, or perhaps Professor Harris had his tongue in his cheek. If the latter, this would account for the many inaccuracies in his paper, such as saying that I used infrared photography—which I did not.

toward the heart. The general arrangement is longitudinal (Fig. 5 and Plate 1, 2). The cranial dural sinuses are the upper terminus; the lower terminus is the network about the caudal vertebrae. The volume capacity of this network is large, much larger than required to return the blood brought in by the arteries.

According to location, the network can be conveniently divided into three intercommunicating divisions (Fig. 4): (1) The central or internal network consists of the vessels which surround the spinal dura mater within the spinal canal. (2) The next group surrounds the first and comprises the vessels within the bone of the vertebrae themselves. (3) There is a plexus external to the vertebral column and surrounding it (Plate 1, 3). The internal peridural network is by far the largest. It forms, in man, an almost continuous meshwork around the dura mater lining the adjacent bones. Except in the upper cervical region, this network is interrupted at regular intervals. Posteriorly there are no veins in the spaces between adjacent vertebrae nor anteriorly, where the intervertebral disks bulge into the spinal canal (Fig. 4). These openings in the plexus give the anterior and posterior halves a ladder-like appearance. Both the longitudinal and the transverse elements of the anterior half are much larger and more regular than those of the posterior half. The absence of the network between the vertebrae posteriorly makes it possible to perform a spinal puncture with little chance of entering the veins. The anthropomorphists might call this a wise provision of nature.

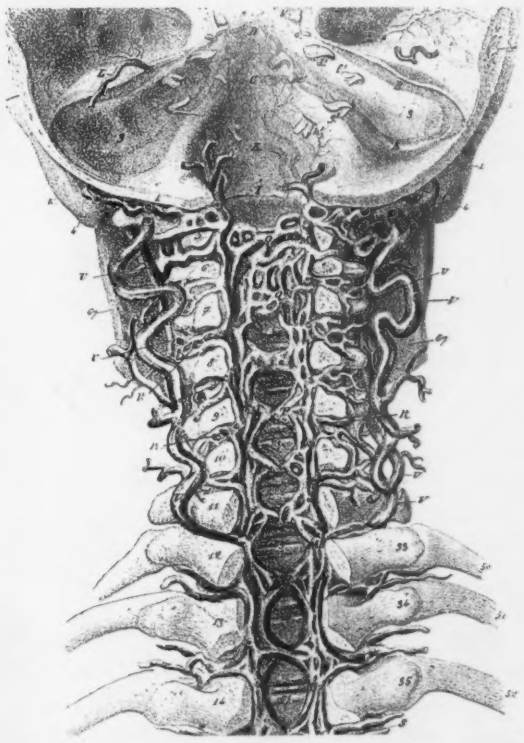

Fig. 5. From Breschet, 1828, like Figure 4. The extradural anterior sinuses are paired and plexiform. The cross connections unite with the large bone emissaries in the middle of the vertebral bodies. The paired sinuses become a network near the skull.

Within the bone of the vertebrae, the veins form a continuous network which joins the veins around the dura mater at many points (Plate 1, 2). Especially at the posterior mid-point of the body of the vertebrae, the connections are large and numerous. From here veins radiate to the anterior equator of the vertebrae (Plate 1, 1). Some of these channels are so large and so direct that they must be considered emissary veins rather than nutrient veins draining the bone.

The posterior network around the vertebrae receives blood from the deep back muscles. It also has longitudinal connections. It unites with the network within the bones and with the segmental veins. The anterior network on the bodies of the vertebrae receives the drainage from the bone itself and the blood from the emissaries as stated and joins with the segmental veins. In the abdomen, the segmental lumbar veins enter both the ascending lumbar veins and the inferior vena cava. Here and there a large emissary vessel leaves a vertebral body and enters the inferior vena cava directly (Plate 1, 2). In the thoracic region, the segmental intercostal veins unite with the superior vena cava through the intermediate azygos veins.

In brief, the vertebral veins are a valveless plexiform network with a longitudinal pattern. They parallel and communicate

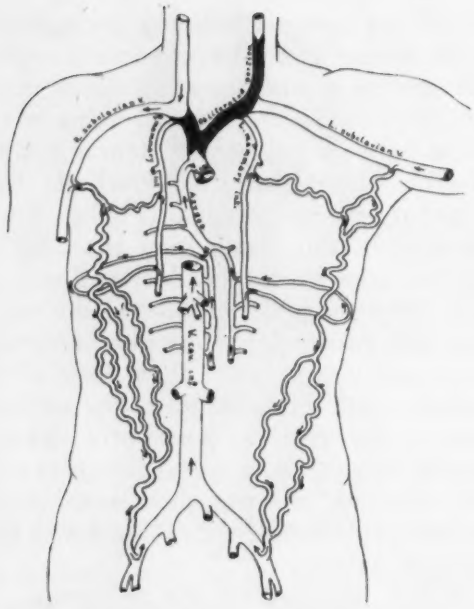

Fig. 6. Illustration used by Prof. Osler shows how the existence of the vertebral veins was forgotten. It was known in the 1830's that the vertebral veins were the principal immediate collateral pathway.

with the superior and the inferior venae cavae. The plexus extends the entire length of the vertebral column and finds a cranial terminus in the dural sinuses. The capacity of the vertebral veins is much greater than required to return blood brought in by the arteries.

This then is the morphologic story. The vertebral veins were first noted in the sixteenth century. Knowledge about them slowly accumulated until the time of Vieussens. After Vieussens they were largely forgotten until Chaussier listed the vessels by name. Breschet, by his methods of injection, by the eloquence of his descriptions, and by the beauty and accuracy of his anatomic plates, presented the first adequate and complete study of the vertebral veins.

It is interesting that three of the four men responsible for the development of this knowledge were at the competitive assembly in 1819. Chaussier and Dupuytren were on the jury and Breschet was a candidate. In 1834, Cruveilhier,[22] a younger colleague of Breschet, commented on the role of these veins when the inferior vena cava was blocked. He also described their significance after the experimental ligation of the jugular veins of dogs.

After Breschet, while the major vessels were described and illustrated by some authors, the knowledge of these veins was scarcely extended. With the pressure to condense the bulk of anatomic knowledge, many anatomists omitted them. In the first four decades of the last century, our knowledge of the vertebral veins was developed and then almost forgotten.

Breschet's comments about the omission of these rachidian veins by the better anatomists of his day could have been rewritten one-hundred years later. They were so unknown that the well-informed late Professor William Osler[47] was unaware of their existence. In a detailed report of a case of thrombosis of the superior vena cava, Professor Osler published a diagram (Fig. 6) showing the supposed routes of the collateral circulation. The vertebral veins are omitted. This figure has been copied with and without credit in describing other cases of caval thrombosis. Today, roentgen phlebographic studies show the extensive part played by the vertebral veins in providing immediate collateral channels after obstruction of either the superior or inferior vena cava. This lack of knowledge and interest was no doubt due to the failure to appreciate their function.

PERSONAL EARLY STUDIES

With the roentgen materials available in the early 1920's, the diploic veins of the skull were not seen on the roentgenograms unless the veins were quite large. I became interested in them and in 1926 I asked Proferssor Artur Schüller,[55] then a visitor in my laboratory in Cincinnati, if he could give me any leads to significant literature. His reply was, "Read Breschet, nothing significant has been done since." Had I asked him about spinal veins, the answer would have been the same. The diploic veins, of course, led me to the vertebral veins. Further interest was provided by the otologists' ligation of the jugular vein which

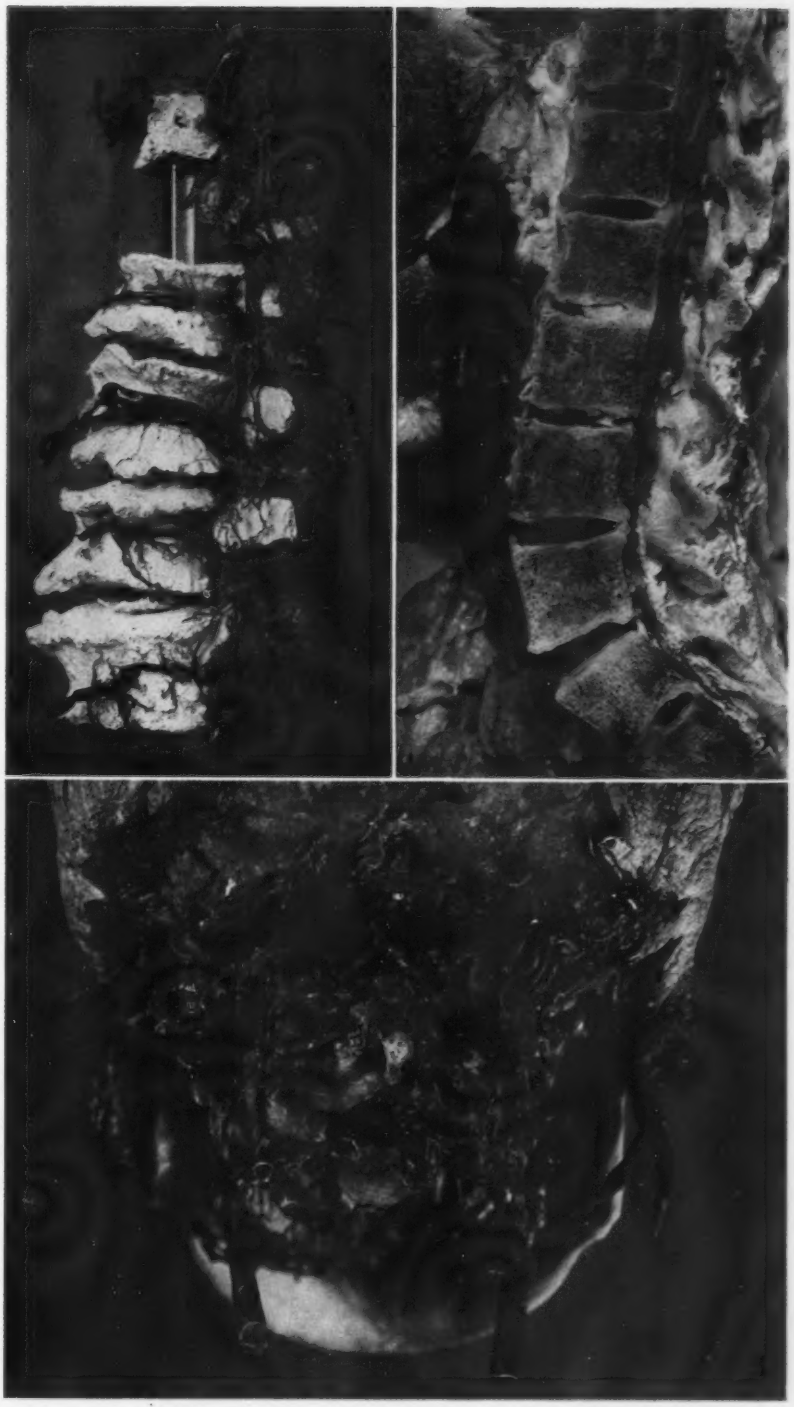

PLATE I. Author's preparations of vertebral veins. (1) Corrosion specimen, human, shows the rich network around the vertebral column. (2) The extradural vertical network, and the extensive intraosseous net. An emissary joins the inferior vena cava. (3) Base of the skull from behind. The posterior extravertebral plexus. Two vertical channels are seen within the spinal canal. (The use of a hand magnifying glass is suggested. Even though it shows the color-screen pattern, it will bring out details that escape the unaided eye.)

was a common procedure before Year One of the Sulfanilamide Era.

In 1927, I began making corrosion preparations of the veins of the head and the neck. At one time I made seven such adult corrosion preparations. The material then used proved to be somewhat temporary, although with a better understanding of how to maintain them, I still have two preparations made in 1930. These specimens showed the extent of the vertebral veins and made self evident their importance in the venous drainage of the head and the neck.

Corrosion studies were supplemented by roentgenograms of cadavera after the injection of roentgenopaque materials.

Fig. 7. Illustration from Batson, 1940. This shows the extensive spread from an injection in the deep dorsal vein of the penis into the vertebral system. (By permission of the J. B. Lippincott Company.)

METASTASES AND AN UNRECOGNIZED VEIN SYSTEM

In 1940, although an occasional suggestion had been made of function, the group of vertebral veins was no more than a morphologic curiosity.

A few papers appeared, as for example Dräger's[25] on the veins in the dog, but in general they were ignored. With my familiarity with Breschet's work, and my study of corrosion specimens, it can be seen why I was able to see in the vertebral veins and their intrapelvic communications, a parallel to the distribution pattern of metastatic carcinoma of the prostate. Pathologists were realizing that metastases could be borne by veins. These veins furnished the only anatomic pattern that coincided with the distribution of the prostatic metastases.

In order to show the venous pattern on roentgenograms, I entered the prostatic plexus of veins by injecting through the deep dorsal vein of the penis. Since this vein drains directly into the prostatic plexus, it seemed to me to be an easy and logical way of access. I have since found that Morel and Duval[45] in their "Manuel de l'Anatomiste," suggest this access for injecting the prostatic plexus of veins. This work has long been on my shelves and perhaps I picked up the idea there and forgot its source. Neither these authors nor Kiss,[36,37] who much later used the same injection, describe the course from here to the vertebral veins.

Our first injection, using radiopaque material of fairly high viscosity, showed a few of the vessels. When the viscosity was reduced so that the injected mass would flow readily into the small vessels, the obtained roentgenogram was all that could be desired (Fig. 7). The vertebral veins and the bone veins of the spine and pelvis were richly injected. The cadaver was in the dorsal recumbent position, and since the vena cava is at a higher hydrostatic level in this position, most of the material remained in the spinal veins. Increasing the amount injected, the material spread as far as the cranial veins. Of special interest to me was the fact that the venae vasorum of the thigh were also filled while the valves in the femoral vein held back the fluid from the large thigh vessels. As noted in the first published report:[6] "The parallelism between the architecture of the injected venous network and the pattern of distribution

Fig. 8. Injection of living monkey with abdominal compression. Material flows up the vertebral veins and even out into an intercostal vein. (By permission of the J. B. Lippincott Company.)

of metastases brings up the entire problem of the mechanism of tumor spread."

While studying these communications, I received a latter from Dr. O'Toole,[48] a former student, asking me if I knew of any anatomic pathway to explain a malignant metastasis to the left frontal sinus from a primary tumor in the right breast. I wrote to Dr. O'Toole that I knew of no such pathway. Less than a week later, with the fluoroscope, I had watched a roentgenopaque mass injected into a small venule in the breast travel by way of the superficial breast veins along the body wall through the axillary and cervical veins. Some of the mass went into the superior vena cava, but much more interesting, the mass ascended along the vertebral veins into the blood sinuses of the cranium. Here was a possible answer to Dr. O'Toole's question. When I made the injection, I was sure that the material would flow upwards along the vertebral veins, yet when I received the inquiry, the answer had been so obvious that it eluded me. The almost universal connections of this extra-cavity valveless network was slowly appreciated.

In experimenting with living monkeys, I reported as follows: "In order to, in part, simulate conditions of increased intra-abdominal pressure such as produced in coughing or straining, a towel was tied about the monkey's abdomen and an injection was again made. Under this condition, while some of the material entered the inferior vena cava, it ascended only part way; some of the thorium medium passed into the vertebral system of veins and can be followed in the roentgenogram past the zone of compression into the vessels of the thoracic spine and out into the lower intercostal veins (Fig. 8). Here then, in the living animal under simulated physiologic conditions, we have a flow which parallels the injection made in the human cadaver, and one which duplicates the pattern of prostatic carcinoma spread." In summary, "The injections in the live animal were not essentially different from those obtained in cadavera, and show that the cadaver injections were not artifacts."

Cranial air emboli as a serious complication after diagnostic perirenal injection of air and after spontaneous and artificial pneumothorax result from air entering the vertebral vein system and traveling to the vertex. Here the air bubble occludes the cerebral veins. The precise symptoms depend upon the amount of air present and its location.

It was stated in the first paper that this problem of venous air embolism was being studied in experimental animals. This incomplete study is here reported in brief. With Drs. Webster, McDonald and Lewey it was found that 5 cc. of air injected into the carotid artery of a dog would kill the dog. In a large dog, 50 cc. of air would embarrass the circulation when injected into a leg vein, but the dog could survive. When

we injected 10 cc. of air into a deep tail vein in the dog (actually a vertebral vein) no ill effects were noted and Dr. McDonald saw no changes in the eye grounds. Upon lifting the head of the anesthetized dog so that it became uppermost, Dr. McDonald observed the retinae to immediately blanche. When tested a few days later, these dogs had altered reflexes. On examining the brains microscopically, Dr. Lewey found pre-existing encephalitis. We had not tested the reflexes of the dogs before the air injection and we felt that our findings should be repeated. Drs. McDonald and Lewey very shortly entered military service and the experiments have not been repeated. There can be no question of the immediate and dramatic blanching of the retina when the dogs were so positioned that the air could ascend cranialward. The role of the vertebral veins in these cases of cranial air embolism has been universally accepted.

It should be appreciated that only rarely have metastases been seen in the state of being born. The usual story of the events in tumor spread is based upon assumptions which appear to be logical, however other assumptions might be made. I suggested that "it is possible to explain most cases of aberrant malignant metastases, aberrant pyogenic metastases and aberrant embolism following air injections by the demonstrated role of the vertebral vein system."

On the basis of the planned experiments and on the basis of these numerous routine injections, the physiologic concept of the vertebral vein system was derived.

I classified the veins of the human body into two principal groups: (1) Those within the pressure chamber of the thoraco-abdominal cavity, and (2) those outside of this cavity.

Within the cavity there are the caval, the pulmonary, the portal and the lumbo-azygos veins. The veins of the extremities which possess valves are part of the caval system. The lumbo-azygos veins are commonly grouped with the caval veins.

Lying outside of this cavity, in contrast to these, are the veins of the head and neck, the veins of the body wall and the valveless veins of the extremities. These vessels, mutually continuous with the veins of the vertebral column (the true vertebral veins), form a separate and distinct group of vessels. This group I have called the "vertebral vein system." This system parallels, joins and at the same time by-passes the cavity veins. It unites the superior vena cava to the inferior vena cava, like the azygos veins, but outside of the pressure cavity.

The vertebral veins, in the strict sense, those veins in and about the vertebral column, form the central part of this extracavity system. This central group provides the connections to the intracavity vessels through segmental vessels and through the large emissary veins in the bodies of the vertebrae.

This concept, so obvious when stated, terminated the existence of the vertebral veins as a morphologic curiosity and placed them in a very active gross physiologic role.

Coughing, or lifting with the upper extremity, not only prevents blood from entering the thoraco-abdominal cavity, it is actually squeezed out of the cavity. The skin of the body wall, including the breast, has many veins without valves. The direction of flow in these vessels is largely a matter of chance. The periosteal veins of the sternum, rib and shoulder girdle, and of course the venous sinuses within these bones are likewise without valves. It would be expected that tumors and abscesses in any of these structures having access to this valveless network could be distributed anywhere along this network. Of course, the ultimate directional flow is toward the heart and the secondary lesions could be in the lung. The point is that they need not be in the lung since the vertebral vein system bypasses the portal, the caval, and the pulmonary systems. The possibility of distribution from the lung without re-entering the heart must also be considered. Cranial metastases from bronchogenic carcinoma

are well known and lung abscesses proverbially have satellite abscesses in the brain.

The interest in metastatic spread should not obscure the function of these vessels in our normal physiology.

In my early enthusiasm, I showed my films to the late Dr. Alfred Stengel, Professor of Medicine. Dr. Stengel asked the question, "What is the normal use of these veins and their connections?" I explained about the function during straining, as in lifting or in physiologic compression. Dr. Stengel's immediate reply was, "Of course, why hasn't this occurred to all of us before?" I had been asking myself that question for some weeks. For one thing, I suppose the answer was too obvious. For the other, we had been told for years that the veins are like the arteries, except that here and there there is a doubling as in the extremities and there is an occasional vein that does not accompany an artery.

Following the publication of the first paper,* there were editorial comments in American, British and Australian journals. Reports began to appear in which unusual findings were explained by the recognition of this vertebral vein system.

For example, J. Purdon Martin[42] suggested that cerebral venous thrombosis following childbirth was due to emboli traveling upward through the vertebral vein system.

In my second paper,[8] the discussion was extended and other accumulated examples of the wide communication of the spinal veins were recorded.

A third paper[7] read before the American Roentgen Ray Society summarized the status of work to that time.

A Spanish translation[9] of the first paper was published in Mexico and Dr. Rahausen[53] in Chile abstracted the third paper in Spanish with extensive translated quotations.

I quoted Hamilton *et al.*[32] about the influence of the veins on spinal fluid pressure. In 1941, Haakon Reitan,[54] of Oslo prepared a paper which, because of war conditions, was read for him in his absence. This was published the same year. This study, made without knowledge of my work, gave beautiful confirmation to parts of it. Dr. Reitan saw a lengthening and narrowing of the intradural iodized oil column as the patient inspired in normal respiration (Fig. 9 and 10). This movement narrowed the column of opaque fluid enough to cause a filling defect to disappear. The beauty of this observation is that it shows that the ebb and flow in the vertebral vein system occurs in normal quiet breathing. It showed indirectly in man what I had shown by injecting veins in the monkey.

From this point forward there has been an increasing number of papers on the vertebral vein system. The anatomic texts are including a discussion of them where none was presented before. In the edition of his anatomy text following my first paper, my friend Professor J. C. B. Grant[31] gave a short but adequate description of the spinal venous complex and described my injection experiments.

The spread of injections in the cadaver

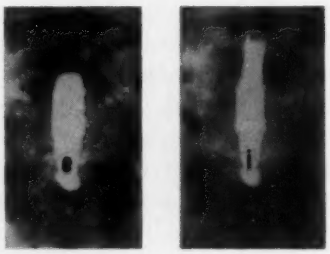

FIG. 9. Reitan's demonstration of compression of the dural sac due to the squeezing by the epidural vein plexus. e, expiration; i, inspiration. (By permission of *Acta radiologica*, and the author.)

* In the publication of the first paper, I had a very fortunate experience. I had sent the manuscript to the editor of a national medical journal. The editor rejected the manuscript, but yet added that he would like to know when it was published because he wished to mention it editorially. With this letter, I talked to Dr. Walter Estell Lee, who was then editor of the *Annals of Surgery*. He asked me to let him have my manuscript. Two days later I was told that the paper had been presented at a meeting of the Editorial Board and immediate publication had been voted. Through the cooperation of Mr. Ellis Bacon and Mr. Walter Kahoe of the Medical Department of the J. B. Lippincott Company, the technical matter of type setting, engraving, etc., were expedited and the paper was in print three weeks after the manuscript had been accepted. Further, the *Annals of Surgery* called special attention to the article. This unquestionably speeded its consideration by the profession.

was studied by Norgore,[46] Anderson,[2] and others. Occasionally some difficulty was encountered, but the injections have been repeated so often that we must conclude that any failure to duplicate the results that I described must be due to some unrecognized technical difficulty. In this laboratory, the injection of the deep dorsal vein of the penis with a color mass has been a routine procedure since our first experiments. Our technical staff has no more difficulty obtaining a good injection than would be expected in any injection.

Looking back over the sixteen years since the first publication of the concept of a physiologic vertebral vein system, the numerous reported studies fall into groups.

HUMAN ANATOMY STUDIES

Several studies of the detailed relations of veins in human antomy have appeared. Especially to be mentioned are the papers from Anson's[4,5] laboratory. These workers studied the minute anatomy of the pararenal veins and the relations of the vertebral veins in a significant number of specimens. With interest in the concept that the vertebral vein system is a pathway for the spread of metastases, they have demonstrated the differences between the left and the right pararenal veins. The veins on the left side are joined intimately with the vertebral vein system. This is in harmony with the well known tendency of metastases from the left suprarenal gland to travel to the brain.

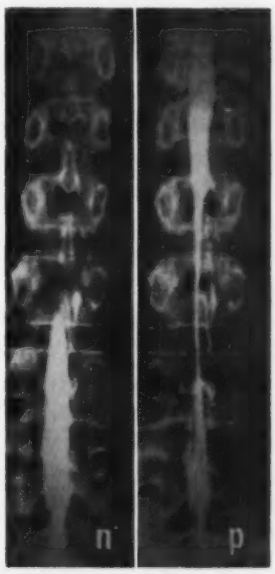

Fig. 10. Another one of Reitan's cases. n, normal level; p, with slight abdominal pressure. (By permission of *Acta radiologica*, and the author.)

Long and Black[41] say that "hypernephromas are notorious for recurring in the lumbar vertebrae, where the cells may frequently be seen as tumor thrombi within the vertebral veins."

At one time the details of anatomy were pursued after the manner of a collector of objects to be treasured in a collection. Today, the focus is toward details which may clarify some phenomena not obvious on the basis of the known facts. Since the vertebral veins were ignored for so long, additional studies clarifying the interrelationships in certain regions may be expected.

Another variety of anatomic study is represented by the extensive paper of Herlihy.[35] Dr. Herlihy is amazed that the vertebral veins themselves have been so long and so completely ignored. He has summarized the history of these vessels since the time of Breschet and is quick to note that the intervening period has added little. He accepts the concept of the vertebral vein system completely and even goes so far as to use a classification in which the veins of the body are divided into two groups: the vertebral veins on the one hand, and all the veins within the body cavities on the other (Fig. 11). This device has the merit of emphasizing the significance of the vertebral vein system and if a diagram must be presented in its simplest form, the Herlihy diagram is excellent. Perhaps it might be well to indicate the subdivisions of the intracavity vessels. I am grateful to Dr. Herlihy for his complete acceptance of this concept and for the beautiful way in which he has marshaled the evidence in its favor.

Lachman[38] has summarized the evidence for a vertebral vein system in a paper which is more general in character but, nevertheless, one built around the concepts of the anatomist.

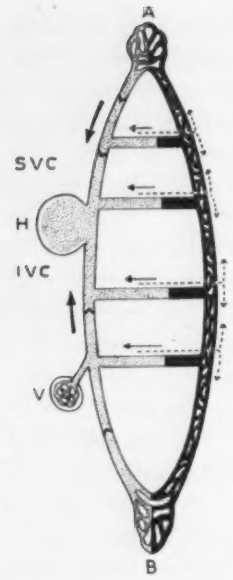

Fig. 11. Herlihy's figure. The valveless vertebral vein system is on the right in black; all of the other body veins are on the left. The middle arrows could have been made double headed. (By permission of the *Medical Journal of Australia*, and the estate of the author.)

Worthman,[68,69] speaking about caval ligation, says: "Since the revelation by Batson of the adequacy of the collateral circulation of the body's axis, many large venous trunks are purposefully ligated. Inferior vena caval ligation was previously done only as a result of accidental injury to this vein during the course of abdominal operation. Walters and Priestly considered such a ligation as a last resort measure . . . more than one hundred cases of caval ligation as a palliative measure in cardiac decompensation are reported." The many surgeons who have seen my specimen have been greatly impressed. Several years ago, Dr. Donald Guthrie said to me, "Since I saw your specimens last year, I have had three cases in which ligation of the inferior vena cava was indicated. On the basis of your demonstration, I had no hesitancy in carrying out the ligation, and the patients had no postligation difficulties."

It is true that the jugular veins were ligated bilaterally sometimes for sinus thrombosis complicating middle ear disease. Also, occasional ligations of the inferior vena cava had been performed. It was known that collateral channels did exist, although erroneous channels were assumed. I have had the opportunity to study a case of acute total occlusion of the superior vena cava and the vena azygos major. This patient, while greatly limited in his activities, survived. During the first year after the occlusion, he showed no abnormal subcutaneous veins. He was accustomed to handling heavy freight in the trucking industry. It must be presumed that due to straining, he used the vertebral system bypass more than the average worker. His vertebral vein system was able to handle the entire venous drainage from the head and neck and both upper extremities in an emergency. Eighteen months after the onset of the occlusion, dilated superficial veins began to appear. The man was more comfortable and was able to increase his activities. However, let it be emphasized that the vertebral vein system was able, in the emergency, to take over immediatly.

There are no statistics available, but I believe that clinical experience shows that bilateral deep neck dissection for removal of malignant lymph nodes is not followed by edema of the head when the patients are laborers accustomed to straining. I assume that this may be true for those who play certain wind instruments, and perhaps even for the chronically constipated individuals.

Studies such as those carried out by Anson and his staff[4,5] will help set the level at which vena cava ligation can be done with safety. It is with full appreciation of the value of antibiotics following ligation that the above statements are made. A competent knowledge of the vertebral vein system removes the fear of ligation. The use of antibiotics allows the ligature to be placed at levels which even with a well developed vertebral vein system would seem hazardous. The whole story of the ligation of the caval veins is presently being rewritten.

The urologists were among the first to

provide instances of the significance of the vertebral veins. Deming and Zaff[23] reported three cases of acute metastatic vertebral osteomyelitis complicating prostatic surgery. They believed that the infection was transmitted to the vertebral column along the veins entering the vertebral vein system. They advised a thorough urinary tract study for infection of all patients with spinal osteomyelitis.

I believe that de la Peña[49] was the first to perform in man the experiment that I did on the monkey. He saw the possibility of using the method in diagnosis. With his junior colleague Amselem,[50] he reported on several of his studies. They feel that they have evidence for the spread of tumors over these veins.

Ciccarelli[18] also demonstrated these vessels in the living human. In this country, Beneventi and Noback,[10] on the basis of my work, studied the pelvic veins by venography in the cadaver. Their studies seemed to be responsible in interesting urologists in the method. Fitzpatrick and Orr[30] demonstrated the pelvic veins in patients as did Abeshouse and Ruben.[1] A more general approach was made by Anderson[2] who forsaw that if you were not especially interested in the prostatic and pelvic veins, the vertebral vein system could be studied by the injection of other vessels. He described his technique in detail. He showed the difference in pattern produced by abdominal compression. When he injected the cephalic vein in four patients, while the patient executed the Valsalva maneuver, the material "backed up" into all of the transverse cervical, scapular and deep cervical veins, into the jugular veins and the veins around the vertebral column. This carried my experiments one step further since the compression that he used was entirely physiologic.

In brief, the cadaver experiments have been confirmed by numerous workers. The injection experiments on the monkey have been confirmed in the patient and even extended. The ebb and flow of blood into and out of the vertebral vein system under physiologic conditions has been established.

Fig. 12. Willis' figure, 1664, showing the vertebral veins of the dog. Although Willis was the first to show cross connections for the whole column in man, he failed to find them in the dog.

The comparative anatomy of the vertebral veins has been studied in earlier periods. Willis,[66,67] who noted the cross connections in man, failed to see them in the dog. His illustration (Fig. 12) shows two separate vertical columns lying within the spinal canal. Breschet[15] states in his larger work, "I point out that I have never observed in man a regularity of the bone canals as pronounced and as constant as in the mammals that I have studied, and I cite especially the carnivora, the plantigrada, the rodentia, the ruminantia, the pachydermata, the solidungula, the cetacea, etc.

"In some of the genera of the various orders of animals, I have seen the wall of the canal covered by a lamina of compact bony tissue which separates and very distinctly isolates the vein from the cancellous substance of the body of the vertebrae."

Cruveilhier[22] performed ligation of the jugular vein in dogs. As in his clinical experience, the vertebral veins acted as the bypassing channels. Dräger,[25] mentioned earlier, described rather completely the vertebral veins in the dog. Since my first publication, there has been an awakening to this problem in the comparative forms. Harrison and Tomlinson[34] have correlated their extensive studies on the seal (Fig. 13) with the vertebral vein system in man.

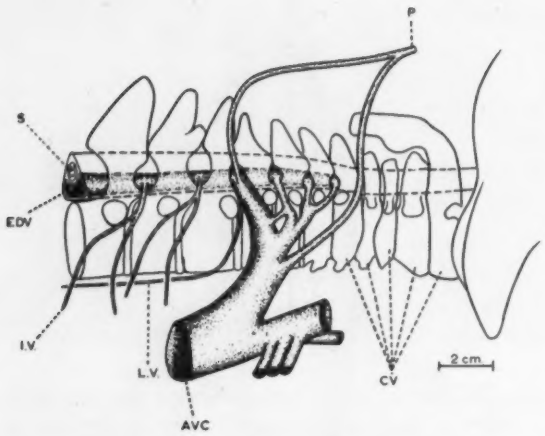

Fig. 13. Harrison and Tomlinson's figure of the enormous extradural vein (EDV) in the porpoise. (By permission of The Zoological Society of London, and the authors.)

From their studies, these veins by their size must be of greater functional significance in the seal. In further work coming from this same laboratory, Tomlinson,[59] accepting the concept of the vertebral vein system, has shown by the use of radioactive NaCl as a tracer that after ligations in dogs there is a flow from the hind limb to the superior sagittal sinus. Bowsher[13] of Liverpool, also accepting the concept, has shown how the bypassing connections increased in size in the rats after jugular ligation. He says that in the rat on the left side, the direct communication between the vertebral venous system and the adrenal gland suggests that this may explain the tendency of tumors of the left adrenal gland to metastasize to the brain.

Recently, Worthman,[68,69] in two careful and detailed papers, reported his studies with the vertebral vein system in the dog. He made numerous experiments to find the direction of flow after ligation of various venous trunks. The direction of flow depends somewhat upon the trunks ligated. These experiments are significant since the venous system is a low pressure system. Minor external pressures and internal swellings may produce results similar to temporary ligation. Worthman began his studies to extend to the domesticated animals the work reported by me. It is evident that with all of the earlier papers being located and summarized and with the mass of new experiments to show the function of the vertebral vein system in the comparative forms, there is little danger of the comparative anatomy of the system falling into oblivion.

When the reports of intraosseous injection of radiopaque material into bone as a diagnostic measure began to appear in the Italian and French literature, we began in the laboratory to make these injections in the cadaver. I found, working with Dr. Goswami, that a needle thrust into the cancerous bone any place in the body could be used as an entrance point into the veins of the bone and hence the deep veins of the part. In this manner we obtained knowledge of many valveless connections with the vertebral vein system.

Dr. Lame[39] has used intraosseous injections to outline venous pathways in patients and has evidence that vertebral osteomyelitis after operations follows these paths.

Now, Fischgold, *et al.*,[29] by placing a trochar in the external occipital protuberance and injecting a radiopaque medium, beautifully outline the lateral sinus, the occipital sinus, the suboccipital veins, and the upper vertebral veins (Fig. 14). This presents a new diagnostic method which permits ready exploration of the veins of the cranium.

In a Hunterian Lecture given at the Royal College of Surgeons, March 14, 1944, J. Leigh Collis,[20] presented his extensive studies of cerebral abscesses complicating thoracic disease. Dr. Collis had repeated a variation of the cadaver injection experiments and showed that the vertebral vein system could be injected as far as the cranium from an intercostal vein. He reviewed the several proposed explanations to account for cerebral abscesses from thoracic disease and felt that transmission over the vertebral vein system was the most probable route. He endeavored to produce cerebral abscesses in cats by injecting into the

azygos vein. He was not able to find any cerebral abscesses in his animals, but one cat developed a mastoiditis. He was disappointed with this series and thought that perhaps the four footed animal was not suitable for this experiment. It seems to me that the production of one mastoid abscess in a series of ten animals is a definite and positive result. It is difficult to obtain, in a healthy animal, the same results that occur complicating an illness. His concise summary might be taken as a model for medical papers. "A careful review has been made of all previous proposals concerning the connection between thoracic disease and cerebral abscesses. Considerable evidence has been given in favor of the view that infected material in the chest is carried to the brain via the spinal veins and that if this material is infected with anaerobic or microaerobic organisms, it is then more likely to result in abscess formation."

In a carefully planned and carefully executed series of experiments, Coman and de Long[21] demonstrated, probably as well as can be demonstrated under experimental conditions, that the vertebral venous system plays a definite role in the dissemination of metastases. The experiments were done upon rats and rabbits. Suspensions of viable tumor cells were injected into the femoral vein while abdominal pressure was momentarily elevated. In the control animals, the tumor growths were uniformly found in the lung. One control animal of the 16 also had tumors in the vertebrae. Of the 14 rats that were injected during brief abdominal compression, 2 had metastases in the lung only, 1 in the vertebrae only and the remaining had metastases in the vertebrae and in the lung. The microscopic studies showed that the growing cells were in the lumina of the vertebral veins. In the control animals, it was noted that the one that had involvement of the vertebrae had squirmed at the time of injection. It is to be interpreted that this squirming elevated the intraabdominal pressure. The experiments in rabbits were equally conclusive. The time period here was too short to allow

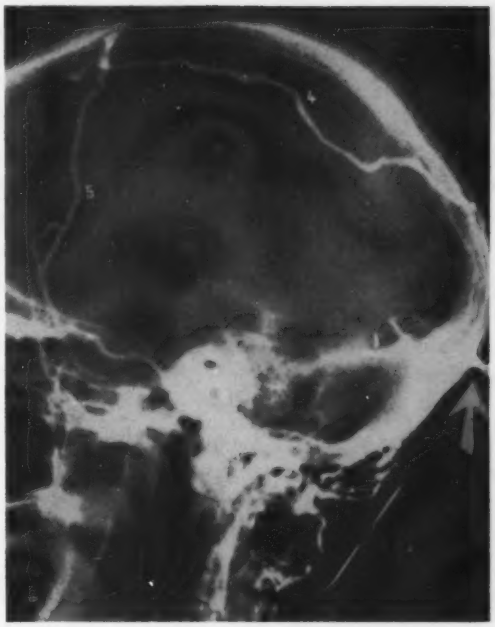

Fig. 14. Injection of the dural sinus, the diploic veins and the vertebral veins in a patient by Fischgold *et al.* The needle was placed in the bone of the external occipital protuberance. (By permission of *La presse médicale*, and the authors.)

the assumption that the vertebral tumors were secondary to the tumors in the lung. These authors conclude, "These findings are in accord with the concept that the frequency of spontaneous tumor metastases to the vertebral column from pelvic tumors in man depends upon the entrance of tumor cell emboli directly into the vertebral venous system, by-passing the lungs." They emphasized that no tumor cell masses were found in arterioles as would have been expected if the arteries had been responsible for the spread. Once it is admitted that carcinoma cells can be transmitted by veins, the conditions of these experiments are more than an approximation of the conditions in man; they are a direct parallel. These experiments showed that the vertebral vein system is an alternate pathway of tumor spread. I know of no series of experiments which demonstrate the isolated involvement of vertebrae by tumor cells passing through the lungs. Coman and de Long[21] established the vertebral vein system as a route of transmission of tumor

cell masses. They have experimentally justified the already wide acceptance of this explanation of spread.

With the numerous new methods of approach to the study of the circulation in man, blood samples have been taken from various parts of the body. To study brain metabolism, blood has been obtained from the jugular bulb. At first, little thought was given to the fact that this might not represent a complete pooling of the cerebral venous blood. In addition to the possible differences between the right and the left side, the blood outflow by way of the vertebral veins must be known. Ferris, *et al.*,[28] on the basis of my studies and variation in their observation, have cautioned workers to consider the vertebral veins and to evaluate their significance. The Air Force Manuals, dealing with the shifting of blood masses with the movements of the body in space, show in all the diagrams the vertebral channels as an alternate outflow from the head. It is doubtful whether this would have been done before 1940. I do not believe that there are any workers in this field of cerbral circulation who do not give full consideration to the vertebral veins as a pathway of egress of blood from the brain case.

Walther,[63] with whom I maintained a correspondence, felt that metastatic lesions could be found in the lung if the search for them was properly carried out. He was very successful in finding a high percentage of these. In his monograph he was willing to concede that metastases could occur by way of the vertebral vein system and included this as a fifth method of metastatic spread.

Willis,[65] who has been unwilling to consider the vertebral vein system as a mechanism for the spread of metastases, now concedes that transmission from vertebra to vertebra may follow this path.

Perey, Lind and Wegelius[51] have shown a massive filling of the central components of the vertebral vein system in infants. This is particularly beautiful when the inferior vena cava is occluded. They also found that, without caval obstruction, material injected into a malleolar vein ascended over the vertebral plexus as well as the vena cava.

The crying infant probably uses his vertebral vein system as much as a laborer. Using the cineroentgenographic method with many exposures per second, Wegelius[64] shows that at the moment of closure of the atrioventricular valve there is a reflux from the atrium back into the azygos vein toward the vertebral connections. Such an ebb and flow has wide implications.

The story of the vertebral vein system is still being written. Many questions remain to be answered. For example, how long can an embolus move about in this complex of veins without going to the heart?

With cineroentgenography coming into full development and with the use of isotopes, such questions should be answerable.

In conclusion, I have had the unusual and gratifying experience of seeing my concept of a vertebral venous system accepted and established. It is today a part of fundamental physiology, and is integrated into clinical practice. I am indebted to Dr. Edward Chamberlain and to the American Roentgen Ray Society for the opportunity to present this summary and I am especially indebted to Dr. Chamberlain for his willingness to accept this very personal topic.

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Photographs and photographic Reproductions by Mr. Alex Vaskelis.

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ROENTGENOLOGIC FOLLOW-UP ON 150 CONSECUTIVE MITRAL COMMISSUROTOMY PATIENTS*

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DESPITE the widespread interest in the medical and surgical aspects of mitral commissurotomy in recent years, there is surprisingly little information in the literature concerning the postoperative changes in heart size and configuration. It therefore appeared worthwhile to review the preoperative and postoperative chest roentgenograms and fluoroscopic findings of the first 150 mitral commissurotomy patients of The Johns Hopkins Hospital. The surgery on this group of patients was performed by Doctor Alfred Blalock and his surgical staff between November, 1949, and June, 1954. The selection of patients with mitral valvular disease for surgery in this series has been previously described.[1] The clinical evaluation of the surgical results in this same group of 150 cases was reported by Milnor.[22]

POSTOPERATIVE CHANGES IN HEART SIZE

Method. Heart size and its changes were evaluated by (1) simple inspection and superimposition of the posteroanterior chest roentgenograms, (2) measurement of the cardiothoracic ratio, (3) measurement of the net frontal plane area using the method of Hodges[16] and Schwarz.[25] No single method of estimating heart size was found completely satisfactory. The least reliable of the three methods appeared to be the cardiothoracic ratio. A change in the frontal plane area of fifteen per cent or more was considered to be a significant change in heart size.

Results. Of the 150 patients, adequate roentgenologic data, including preoperative and postoperative fluoroscopy, posteroanterior, lateral, and oblique chest roentgenograms, were available in 115. Eleven of the remaining 35 patients died within two weeks of surgery, 4 had thoracotomy without completion of the commissurotomy, and 20 had inadequate postoperative roentgenologic studies.

All portable chest roentgenograms were excluded from the study of heart size. Because of this, an adequate evaluation of changes in heart size within the first two postoperative weeks could not be made. The changes in cardiac size from two weeks to five years following surgery are summarized in Table I.

TABLE I

POSTOPERATIVE CHANGES IN HEART SIZE

No. of Cases Studied	103 = 100%	86 = 100%	54 = 100%
	2 wk. to 3 mo.	3 mo. to 5 yr.	1 to 5 yr.
	Per Cent	Per Cent	Per Cent
Unchanged	46	55	54
Decreased	8	26	22
Increased			
Persistent	1	19	24
Transient	45	—	—

In the first three months following surgery, a transient increase in the cardiac silhouette occurred in almost half the cases. A permanent increase or an immediate decrease in size was unusual. Because the silhouette almost always regressed to at least its preoperative size within three months (Fig. 1), the initial increase was considered to be due to postoperative pericardial fluid in most cases. In a few patients, this was confirmed by pericardial tap and injection of air. In other series,[18,28] post-

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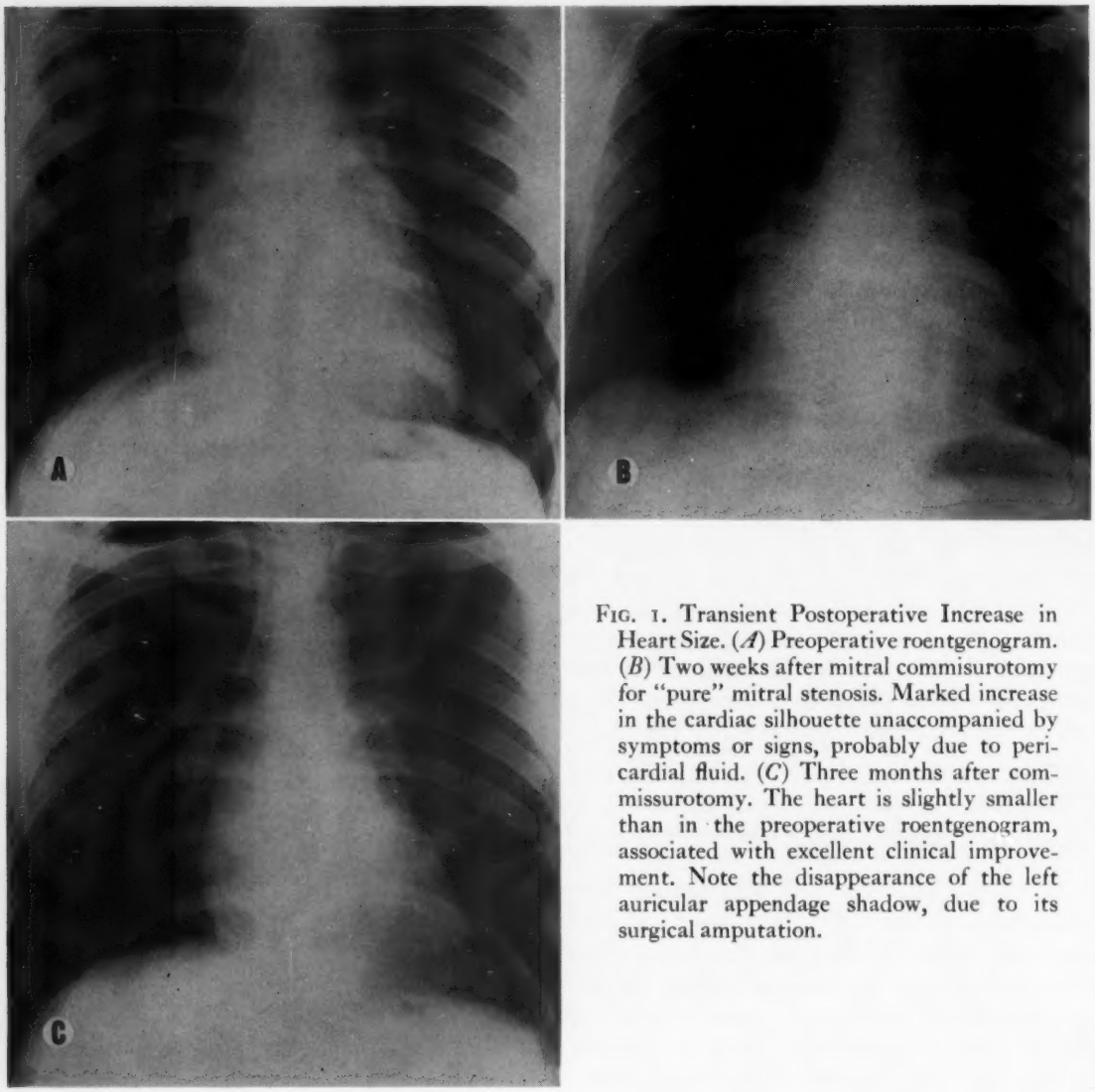

FIG. 1. Transient Postoperative Increase in Heart Size. (*A*) Preoperative roentgenogram. (*B*) Two weeks after mitral commisurotomy for "pure" mitral stenosis. Marked increase in the cardiac silhouette unaccompanied by symptoms or signs, probably due to pericardial fluid. (*C*) Three months after commissurotomy. The heart is slightly smaller than in the preoperative roentgenogram, associated with excellent clinical improvement. Note the disappearance of the left auricular appendage shadow, due to its surgical amputation.

operative pericardial aspirations have revealed serosanguineous fluid, sometimes with clotted blood. Regardless of whether the initial transient increase in heart size was pericardial or myocardial, it was entirely unrelated to the eventual clinical result, the development of rheumatic activity or to the presence of Aschoff bodies in the amputated auricular appendage.

In the majority of patients followed from three months to five years, there was little objective evidence of improvement in heart size, in contrast to the dramatic symptomatic results of surgery (Table 1). Similar observations have been made in other series.[7] The over-all roentgenologic results tended to be somewhat poorer the longer the period of follow-up. Both the clinical and roentgenologic results in the last 100 patients of the series were better than in the first 50 patients. This may have been due to improvement in surgical technique in addition to a shorter follow-up period. Within the relatively narrow age range of twenty to fifty years in this series, age had no apparent effect on changes in heart size. As to be expected, the larger preoperative hearts tended to show more striking de-

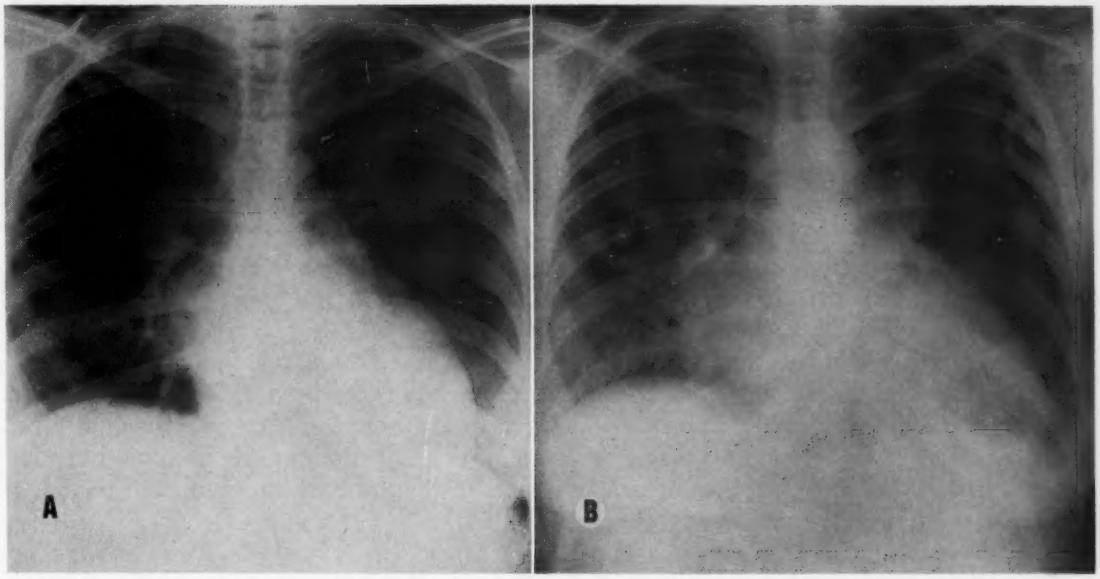

Fig. 2. Late Postcommissurotomy Syndrome. (*A*) Preoperative roentgenogram, showing prominence in the right heart border, due to enlarged left atrium, and a long and prominent left heart border. At surgery, mitral stenosis with some element of insufficiency was found. Good clinical improvement. (*B*) Two years postoperatively, increase in cardiac enlargement and pulmonary congestion developed concomitantly with a reactivation in the rheumatic process.

creases in size than the smaller hearts. In several patients, the preoperative decrease in cardiac size due to medical management was more marked than the postoperative decrease. It is therefore of prime importance in an objective study of the effects of surgery to obtain roentgenograms immediately prior to the operation for future comparison.

Eight patients had no change in heart size following surgery for two or three years, but then developed a definite enlargement. In 4 of these, the enlargement was associated with a late "postcommissurotomy syndrome" (Fig. 2); in 2 others, the enlargement was associated with a normal pregnancy. In the 2 remaining cases, the clinical improvement following surgery continued to be excellent and the cardiac enlargement was unexplained.

Comparison of Roentgenologic and Medical Findings. Allowing for the fact that the roentgenologic improvement was not as great as the subjective improvement, there was fairly good correlation between the roentgenologic and clinical results in most patients (Table II). A minority of patients who were improved had a significant decrease in heart size, while none of the unimproved patients showed such a decrease.

Table II

COMPARISON OF CHANGES IN HEART SIZE WITH CLINICAL RESULTS

Clinical Results	No. of Cases	Per Cent of Group		
		Unchanged	Decreased	Increased
Group A Greatly Improved	57	58	30	12
B Moderately Improved	21	48	24	28
C Unimproved	9	22	0	78
D Late Deaths	6	50	0	50

A postoperative increase in heart size occurred more frequently in the unimproved patients.

The degree of cardiac enlargement preoperatively had only a slight influence on the eventual clinical result (Table III). Even in patients with marked enlargement, surgery produced some benefit in 75 per cent. The percentage of early and late deaths, however, was highest in this group (19 per cent).

Nineteen patients in this series had hearts of normal size and in 6 of these, the cardiac contours were quite normal (Table III). It has been well established that some patients with mitral stenosis and hearts of normal size may have moderate clinical disability,[2] and that evidence of left atrial enlargement may not be demonstrable.[9] This group had excellent clinical results. Twenty-three per cent of this group had a slight increase in heart size postoperatively and only 7 per cent showed a decrease.

The clinical preoperative disability of the patients (graded by the functional classification of the American Heart Association[5] was definitely related to preoperative heart size. This study confirmed previous observations[2] that patients with frank congestive heart failure secondary to mitral disease almost always have some degree of cardiac enlargement. Patients with established auricular fibrillation before surgery had relatively larger hearts (average frontal plane area +54 per cent) than non-fibrillators (average area +39 per cent).

Twenty-four patients (16 per cent of the series) developed the so-called "postcommissurotomy" syndrome, consisting of chest pain, fever, and signs of pleuritis and pericarditis,[7,8] commonest in the second postoperative week.[18] When the syndrome occurred within the first two postoperative months, it was not accompanied by significant roentgenologic changes. Eight patients, however, with a late onset of the syndrome between two months and four years developed a definite increase in heart size at the time of the clinical rheumatic activation.

Changes in Heart Contours Following Surgery. The contour of the left auricular appendage, seen on the posteroanterior chest roentgenogram, became diminished in 57 per cent of the cases, due merely to its surgical amputation. The left atrial contour became diminished in 17 per cent, but increased in 7 per cent. In demonstrating left atrial enlargement by the posterior displacement of the barium-filled esophagus, Jacobson *et al.*[17] found the left lateral position more satisfactory than the right anterior oblique. This was found especially true in this comparative study of roentgenograms before and after surgery because the positioning of the patients for oblique

TABLE III

RELATION OF PREOPERATIVE HEART SIZE AND EVENTUAL CLINICAL RESULT

Frontal Plane Area Per Cent Variation from Normal		Preoperative Heart Size			
		Normal 0–±10%	Slight Enlargement +10 to +39%	Moderate Enlargement +40 to +59%	Marked Enlargement +60% or over
No. of Cases		19=100%	39=100%	29=100%	47=100%
		Per Cent	Per Cent	Per Cent	Per Cent
Clinical Result	A	78	62	59	62
	B	17	20	24	13
	C	5	5	3	6
	early D	0	8	7	11
	late D	0	5	7	8

projection could not be duplicated as readily as for the true lateral projection.

The prominence of the main pulmonary artery decreased in 28 per cent and increased in 12 per cent, and similar changes were observed in the peripheral pulmonary vascularity.

When the heart showed an over-all decrease in size, this was found chiefly due to decrease in the right ventricle.

Correlation of Roentgenologic and Surgical Findings. Of 144 patients in whom sufficient data were available, calcification of the mitral valve was palpated surgically in 47 (33 per cent). In the same group of patients, the mitral valve calcification was demonstrated roentgenologically in only 25 (17.5 per cent). Even if the 3 patients with doubtful calcification and 2 additional patients without preoperative fluoroscopy were excluded, the roentgenologic demonstration of calcification was no better than 60 per cent. One-third of these patients was studied by a single experienced fluoroscopist; the percentage of calcified valves demonstrated by him was no greater than in the series as a whole. The occurrence of calcification in the mitral valve had a slightly adverse effect on the prognosis (Table IV).

In 3 patients, there was calcification of the posterior and left lateral endocardium of the left atrium. The calcification was best demonstrated in the right oblique view, but was also seen on the posteroanterior view, as described by Epstein.[10] In two patients, there was visible calcification of the endocardium of the left auricular appendage, best seen in the posteroanterior view. This was of considerable practical importance, for the calcification was associated with large auricular thrombi which rendered the surgical procedure difficult in one patient and impossible in the other. No pericardial calcifications occurred in this series.

TABLE IV

INFLUENCE OF MITRAL VALVE CALCIFICATION ON PROGNOSIS

No. of Cases		Valvular Calcification		
		None	Found Only at Surgery	Roentgenologically Demonstrated
		93 = 100%	20 = 100%	22 = 100%
		Per Cent	Per Cent	Per Cent
Clinical Result	A	71 } 85	55 } 80	45 } 68
	B	14	25	23
	C	4	10	9
	late D	4	10	9
	early D	7	0	14

Some degree of mitral insufficiency was found at surgery in 30 patients (20 per cent of the series). Eighteen of these had minor insufficiency associated with mitral stenosis. The clinical results in this group were quite good, although only 1 patient had a postoperative decrease in heart size. The other 12 patients had major mitral insufficiency and half of these had no element of stenosis at all (Fig. 3). Only 1 of these patients was apparently improved by the surgical procedure. The roentgenologic follow-up of these 12 patients was so inadequate that no conclusions could be made about changes in heart size.

The average preoperative heart size in the patients with major mitral insufficiency was considerably greater than in the average patient with mitral stenosis alone, whereas a minor degree of insufficiency had apparently no influence on heart size (Table V). The incidence of extreme enlargement of the left atrium was also considerably higher with major insufficiency than with mitral stenosis. This was not a helpful diagnostic criterion, however, because the absolute number of "giant" left atria in stenosis and insufficiency in this series was about the same. The incidence of mitral calcification in the patients with mitral insufficiency was higher (43 per cent) than in cases with pure stenosis (10 per cent), as has been reported by others.[2,23,29]

In the patients with pure mitral stenosis, the correlation between the severity of the stenosis, as estimated by the surgeon's ex-

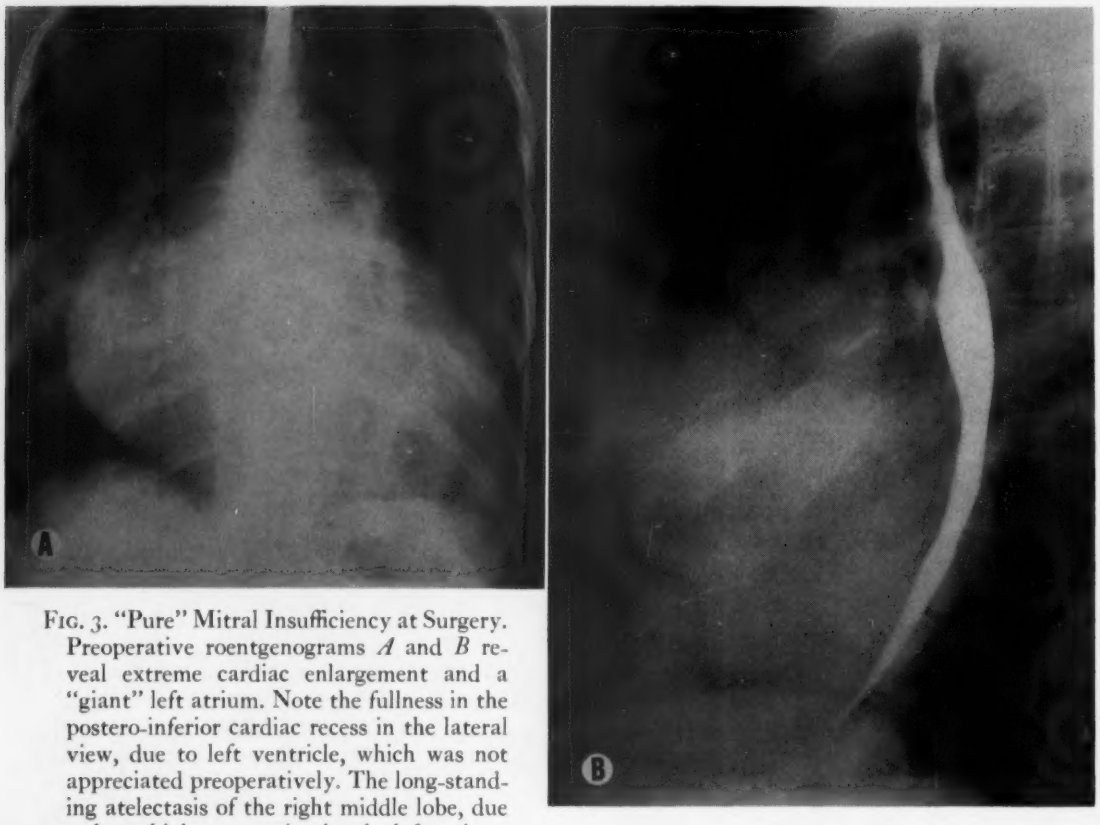

FIG. 3. "Pure" Mitral Insufficiency at Surgery. Preoperative roentgenograms *A* and *B* reveal extreme cardiac enlargement and a "giant" left atrium. Note the fullness in the postero-inferior cardiac recess in the lateral view, due to left ventricle, which was not appreciated preoperatively. The long-standing atelectasis of the right middle lobe, due to bronchial compression by the left atrium, was verified at postmortem.

ploring finger, and the heart size, as measured on the preoperative roentgenograms, was not marked. The average frontal plane area in severe stenosis was about 10 per cent greater than the average area in cases with slight stenosis, and 5 per cent greater than in those with moderate stenosis. The degree of dilatation of the mitral valve produced at surgery, usually expressed as one, one and one half or two fingerbreadths, had no apparent influence on either the clinical results or on the postoperative cardiac size.

TABLE V

INFLUENCE OF DEGREE OF REGURGITATION ON SIZE OF HEART AND LEFT ATRIUM

	No. Cases	Av. Heart Size	Per Cent with Very Large Left Atrium
Pure Stenosis	95	+47%	11
Minor Insufficiency	18	+35%	6
Major Insufficiency	12	+79%	70

The size of the main pulmonary artery estimated roentgenologically from 1+ to 4+ corresponded quite well with the surgeon's descriptions. The correlation between the size of the left auricular appendage shadow corresponded fairly well with the size estimated at surgery. In a few patients with an extremely small auricular appendage, commissurotomy was technically difficult; its small size could usually be determined from the posteroanterior chest roentgenogram. There was absolutely no relation between the size of the appendage and the presence or absence of thrombus material within it.

Pulmonary Changes Associated with Mitral Valvular Disease. An almost con-

secutive series of 50 patients had cardiac catheterization before surgery, and adequate roentgenograms were available in 46 of these. The presence of pulmonary vascular changes, with dilatation of the hilar vessels and narrowing of the peripheral vessels (as described by Davies *et al.*[6]) correlated fairly well with the level of pulmonary artery pressure. All patients had some degree of pulmonary hypertension. When the vascularity was evaluated as 3+ or 4+, the pulmonary artery pressure was usually 65 mm. mercury systolic and 40 mm. mercury diastolic, or over (Fig. 4). There was poor correlation with the pulmonary capillary pressure and no correlation with the cardiac output index and calculated pulmonary arteriolar resistance. The clinical results following surgery were not related to either the degree of pulmonary hypertension[22] or the prominence of the pulmonary vascularity (Fig. 5).

In pulmonary hypertension accompanying mitral stenosis, the statement has often been made that the heart is usually normal or almost normal in size.[14] In this series, however, no correlation was found between pulmonary hypertension and heart size. Several patients with extreme pulmonary hypertension had marked cardiac enlargement. Patients with a high degree of pulmonary hypertension tended to have a more dramatic improvement in heart size than the average case of mitral stenosis. In patients with a severe degree of mitral stenosis, the roentgenologic changes in the pulmonary vascularity tended to be quite marked. As in the studies of Short,[27] how-

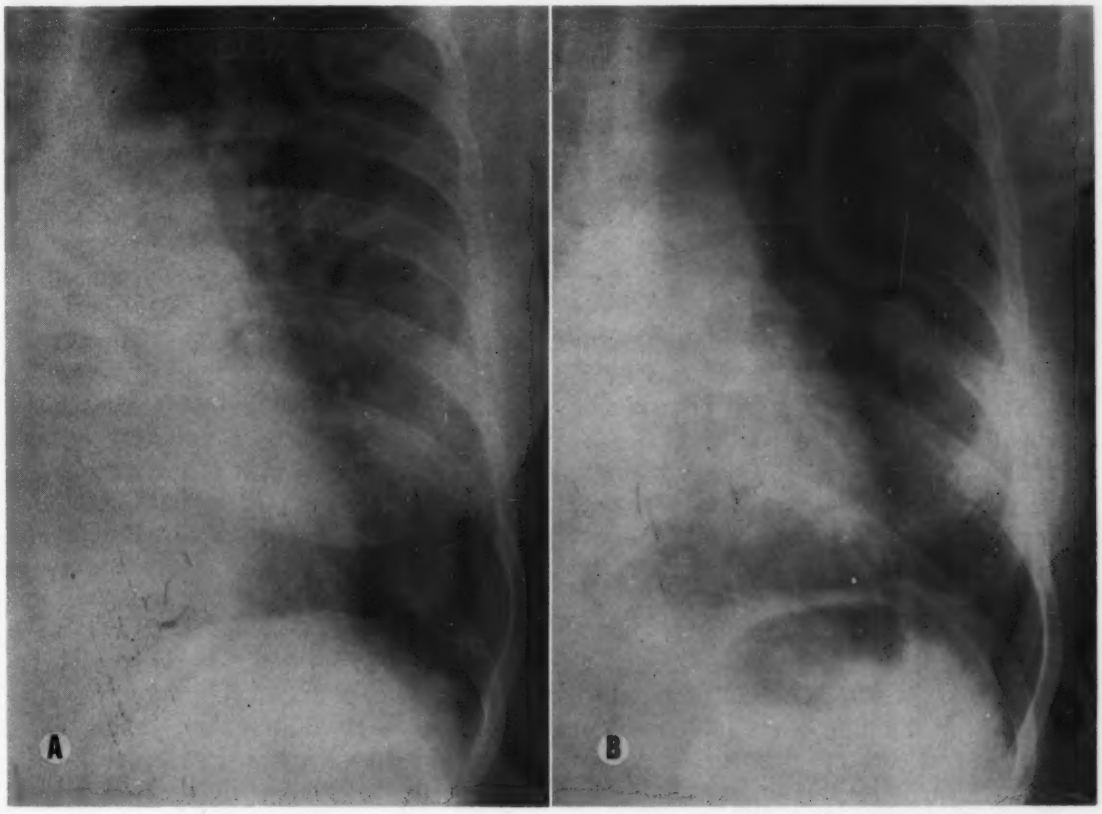

Fig. 4. Extreme Pulmonary Hypertension Secondary to Mitral Stenosis. (*A*) Preoperative roentgenogram. The pulmonary artery segment is prominent, and the hilar vessels are dilated. Definite "B" lines are seen just above the costophrenic angle. (*B*) Six months postoperatively, the main and peripheral pulmonary artery shadows are much less prominent and the "B" lines have disappeared. Small intrapulmonary ossified nodules are now visible. Excellent clinical and roentgenologic improvement.

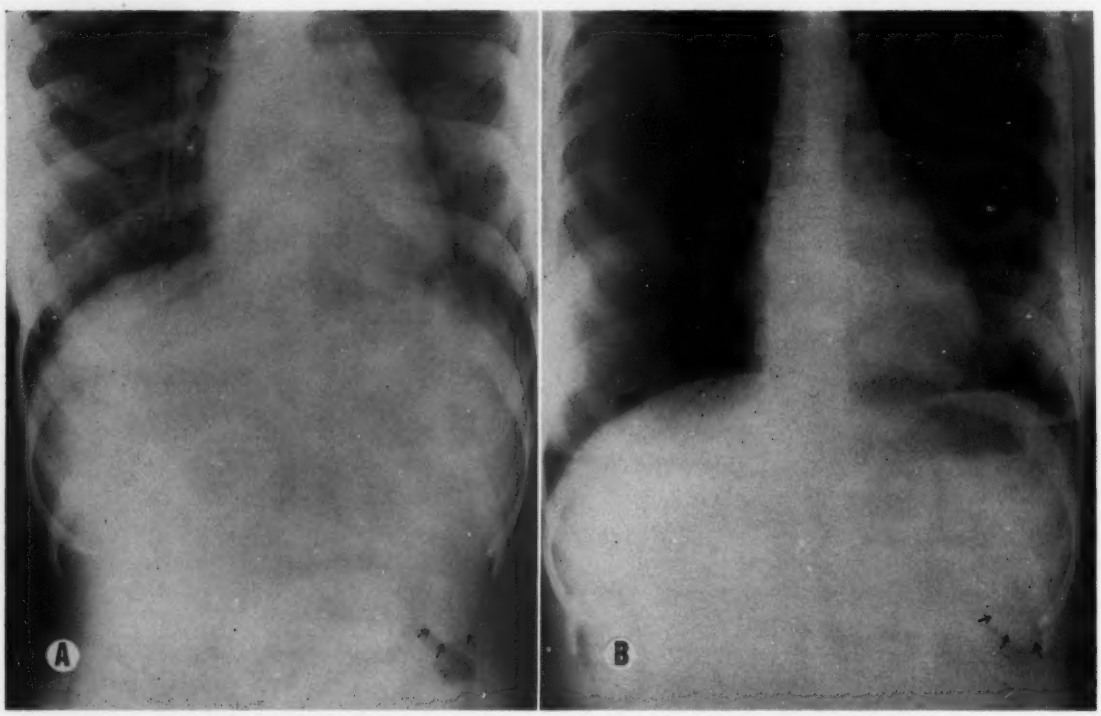

FIG. 5. Mild Pulmonary Hypertension Secondary to Mitral Stenosis with Some Insufficiency. (*A*) Preoperative roentgenogram. The pulmonary vascular changes are minimal. (*B*) Two and one-half years postoperatively. The pulmonary vascularity appears normal. The pulmonary artery and left auricular appendage segments are less prominent. The lower border of the spleen is higher, indicating a postoperative decrease in size. Excellent clinical result.

ever, it was not unusual for 2 cases with an identical degree of mitral stenosis to have marked differences in the degree of hypervascularity and pulmonary hypertension. The explanation for this is not known. The excellent correlation found between the degree of functional clinical disability and the degree of pulmonary vascular change reported by Fleischner and Sagall[13] was not found in this study.

"B" lines, first described by Kerley,[19] have been definitely established histologically as pulmonary interlobular septa.[12] They appear as thin, parallel, horizontal, dense lines in the lung bases extending out to the pleural surfaces and are best seen just above the costophrenic angles usually in the frontal projection, but occasionally in the oblique views.[4,12,15,27,30] In mitral valvular disease, these abnormal lines are thought to be due to edema and lymphatic dilatation in the interlobular septa,[15,30] secondary to pulmonary hypertension. "B" lines were present in 33 per cent of the patients in this series (Fig. 4). In the 46 patients who had had catheterization, the presence of "B" lines did not correlate as well with the level of pulmonary hypertension as the assessment of the pulmonary vascularity alone. When the lines were marked, fairly severe hypertension was invariably present, but a few of the cases with the greatest pulmonary hypertension had no visible lines. When these lines were minimal in extent, they had no correlation with the level of pulmonary hypertension. In the study of Carmichael, Julian, Penrhyn Jones and Wren,[4] the presence of "B" lines correlated well with the pulmonary capillary pressures, but did not in this series. There was, however, a fairly good correlation with the calculated pulmonary arteriolar resistance.

In a series of 134 patients, Grainger and

Hearn[15] found that the clinical results following commissurotomy were considerably better in patients with "B" lines than in those without the lines. They, therefore, considered the presence of the lines a valuable prognostic sign. However, in this series, on comparison of patients with and without "B" lines, the clinical results following surgery were found to be identical. This was to be expected, since the clinical results were not materially affected by the severity of the pulmonary hypertension. The lines were observed in patients with mitral insufficiency as well as in those with mitral stenosis.

Observation of the "B" lines before and after surgery proved valuable, because the disappearance of the lines a few weeks after surgery was often the first objective evidence of improvement. In 24 patients in whom the "B" lines promptly disappeared, the clinical results were invariably satisfactory. On the other hand, in 9 patients in whom the "B" lines persisted postoperatively for several months, the clinical results were rather poor in 6 of these.

Multiple intrapulmonary ossified nodules associated with mitral valvular disease were present in 10 per cent of the patients in this series. These lesions may be confused with scattered calcified nodules of healed histoplasmosis, but they are usually round, more marked in the lung bases and associated with severe pulmonary vascular changes (Fig. 4). Definite "B" lines were usually present. In 5 patients, cardiac catheterization showed severe pulmonary hypertension with pulmonary systolic pressures in excess of 70 mm. mercury. Lung biopsies taken at the time of mitral commissurotomy showed advanced changes of chronic passive congestion. Sometimes biopsy has revealed the nodules, consisting of concentric lamellations of bone.[30]

Only 1 patient in this series had diffuse miliary lesions throughout the lung fields, presumably due to hemosiderosis. Baker, Brock, Campbell, and Wood[2] reported such lesions in 10 per cent of a group of 50 patients. This large number may possibly be attributed to the high incidence of severe pulmonary hypertension observed in their patients. Esposito[11] reported a roentgenologic incidence of 5 per cent in 100 postmortem cases of rheumatic heart disease. Several patients in the present series had miliary-like lesions which cleared promptly after surgery and were therefore not due to hemosiderosis but to pulmonary congestion.

Roesler[24] described bronchial compression and atelectasis secondary to left atrial enlargement, but this entity was denied by Schwedel.[26] In 2 patients of this series, there was persistent atelectasis of the right middle lobe associated with gross left atrial enlargement. Both patients died postoperatively, and at postmortem no explanation was apparent for the atelectasis other than the bronchial compression (Fig. 3).

In a few patients, sudden episodes of hemoptysis have appeared postoperatively associated with localized patches of pulmonary infiltration. It has sometimes been difficult or impossible to tell whether these pulmonary changes were due to infarction or merely to localized hemorrhage.

Changes in the Splenic Outline. In only one-half of the patients was the lower border of the spleen visible on chest roentgenograms. In about one-third of this group, the spleen appeared to be somewhat larger than normal. In several patients, a definite change in splenic size occurred postoperatively, usually paralleling the cardiac changes in size (Fig. 5).

DISCUSSION

The majority of patients in this series showed no significant change in heart size following commissurotomy; about 25 per cent of the group had a significant decrease in size and 20 per cent had a significant increase. It is quite possible that patients without a change in heart size may have developed cardiac enlargement had surgery not been performed. A true assessment of the effects of surgery would be possible only if a similar group of patients could be followed without surgery.

Only 8 of the 150 patients developed a reactivation of the rheumatic process with an increase in heart size postoperatively. These results differ markedly from those of Soloff and Zutuchni,[28] who reported enlargement, presumably due to rheumatic activity, in 11 out of 20 patients six months or more following commissurotomy. Only 2 of our cases developed disproportionate enlargement of the left ventricle and both patients had clinical evidence of mitral insufficiency appearing after surgery. Otto *et al.*[23] found only 1 instance of postoperative left ventricular enlargement in 54 patients. These findings contrast with those of Busch,[3] who found postoperative left ventricular dilatation in the majority of 22 patients.

Roentgenologically, only 60 per cent of mitral valvular calcifications were detected. A relatively large percentage of minimal calcifications of the valve cusps were missed fluoroscopically, as in the series of Baker, Brock, Campbell and Wood.[2] Other authors, however, have demonstrated 80 per cent[23] and 86 per cent[20] of valvular calcifications found at surgery. The presence of mitral valvular calcification in this series made the prognosis slightly worse. In a few instances, calcification of the valve may be so extensive that commissurotomy is rendered technically impossible.[8] The incidence of calcification was higher in mitral insufficiency than in mitral stenosis; the incidence of surgically-created insufficiency may also be increased in the presence of calcification.[23,29]

Twelve patients or 8 per cent of the group had marked mitral insufficiency at surgery, and these represent clinical and rentgenologic diagnostic failures. The inability to differentiate mitral stenosis from insufficiency or to evaluate the relative importance of the two lesions when both are present has remained an unsolved problem of cardiac roentgenology. The fact that minimal insufficiency accompanying stenosis does not seriously alter the surgical results has increased the difficulties in the preoperative assessment of patients. The amount of depression of the cardiac apex in the posteroanterior chest roentgenogram, the fullness of the postero-inferior cardiac recess in the lateral projection, and the relative prominence of the right and left borders in the left oblique projection have sometimes been unreliable signs of left ventricular enlargement. Conversely, several false positive diagnoses of left ventricular enlargement and mitral insufficiency have been made. Systolic expansion of the left atrium demonstrated either fluoroscopically or kymographically was of no help in diagnosis; indeed, the majority of cases with systolic expansion in this series proved to have mitral stenosis. Intravenous angiocardiography has also often failed in the differentiation of mitral stenosis from insufficiency.[21] Therefore, in patients with combined stenosis and insufficiency, surgical exploration is sometimes the only means of evaluating their relative functional significance.[22]

The study of the pulmonary vascularity on the plain chest roentgenograms alone proved to be a reasonably accurate method for estimating pulmonary hypertension, comparable to other reported series.[4,6,13,30] This, however, is of little practical importance either from the viewpoint of prognosis or management.

SUMMARY

1. In a series of 150 consecutive mitral commissurotomies, roentgenologic follow-ups from two weeks to a maximum of five years postoperatively were available in 115 patients.

2. The roentgenologic changes in cardiac size and configuration as a result of the surgery were described.

3. The roentgenologic findings were correlated with the medical, surgical, and cardiac catheterization data.

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INTRAVENOUS CARBON DIOXIDE FOR INTRACARDIAC GAS CONTRAST IN THE ROENTGEN DIAGNOSIS OF PERICARDIAL EFFUSION AND THICKENING*

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THE difficulties in establishing a roentgen diagnosis of pericardial effusion are generally recognized.

Our group has previously demonstrated the safety of intravenous carbon dioxide gas for intracardiac contrast experimentally in anesthetized dogs in doses of 7.5 cubic centimeters per kilogram of body weight, rapidly injected.[2,3] The relatively slight physiologic disturbances resulting from such large doses have been studied.[1]

Adult patients have tolerated smaller doses of intravenous carbon dioxide well. There have been no discomfort and no reaction following rapid injections of 50 to 100 cc. of the gas.

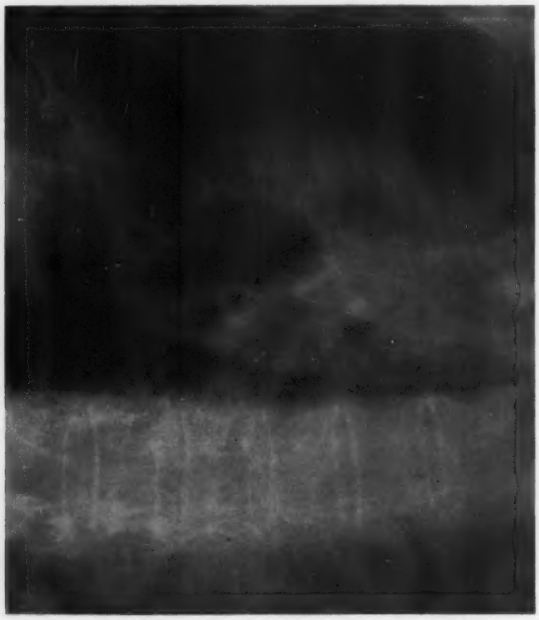

FIG. 1. Carbon dioxide gas in right atrium. Large pericardial effusion in myxedema.

The lateral margin of the right atrium is readily demonstrated by intracardiac gas contrast with the patient in left decubitus using the roentgen beam horizontally (Fig. 1 and 2). The buoyancy of the gas bubble maintains it in contact with the lateral atrial wall until the gas is dissipated. The greater specific gravity of the heart and its blood content relative to any pericardial effusion ensures that fluid present, if free, will appear lateral to and above the visualized right atrial wall.

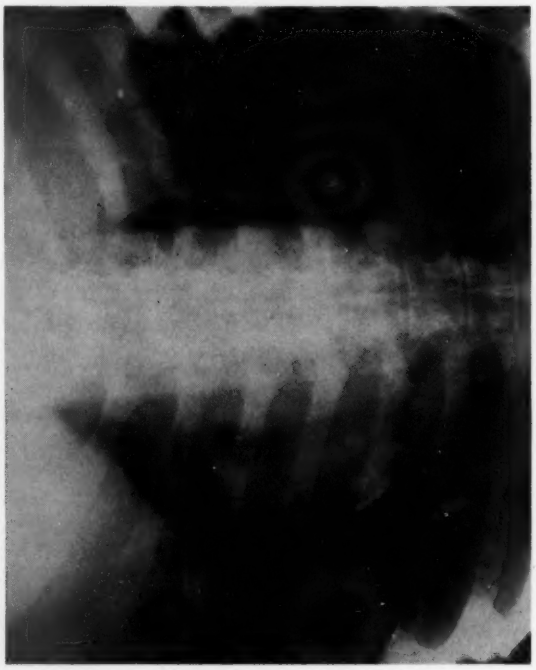

FIG. 2. Thickened pericardium in calcifying constrictive pericarditis visualized between lung and gas in right atrium.

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TECHNIQUE OF INJECTION

A continuous intravenous drip is arranged in the antecubital vein of the dependent arm. A syringe with a 3-way stopcock attached is flushed with saline and checked to eliminate air leak. A short sterile tube is attached to the tank of 100 per cent carbon dioxide. The tube is flushed with carbon dioxide and the syringe is promptly attached, filled with gas and emptied through the side arm of the stopcock. The syringe is refilled with carbon dioxide and the injection is immediately made into the intravenous tubing.

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ABNORMALITIES SEEN ON CHEST PHOTOFLUOROGRAMS AND DIAGNOSABLE HEART DISEASE

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IMPRESSIONS regarding the value of chest photofluorograms in detecting heart disease have been based mainly upon the yield in tuberculosis chest surveys where heart disease was noted only incidentally during the initial screening of the survey films. On the average, cardiovascular abnormalities have been noted in one half of one per cent of these films (ranging from 0.1 to 1.0 per cent) and, where follow-up has been done, these have produced between 1 and 2 confirmed cases of heart disease per thousand examinations, of which 60 to 80 per cent were previously known.[3,5,6,12,15,16] Much higher yields have been reported where particular attention has been paid to cardiovascular shadows in examining the survey films.[8,11] In a community-wide chest photofluorographic survey in Boston, Massachusetts, during a time in which cardiovascular abnormalities were considered to be only incidental findings, the readers noted 0.4 per cent of the films positive for cardiovascular disease.[16] In films which they knew were to be rechecked as part of a cardiovascular disease study, however, they noted 1.1 per cent positive. Special readers in rereading these films, noted *an additional* 2.4 per cent positive, giving a total of 3.5 per cent of the survey films considered positive for some cardiovascular abnormality.[10] The ultimate yield of confirmed heart disease among film rolls which received special reading was 16.2 per thousand examinations as compared with 2.3 per thousand among films read in the usual manner. Less than half (44.5 per cent) of the cases identified on the specially read films were previously known; whereas two thirds of the cases identified on films read in the usual manner were previously known.

There is little doubt that more cases of heart disease could be discovered in routine chest surveys if films were read more carefully for cardiovascular disease. Before cardiovascular shadows receive special attention, however, there are a number of questions that need to be answered. Many patients with heart disease have normal cardiac silhouettes in the posteroanterior view, and it is not known whether even under the most favorable circumstances, a sizable proportion of persons with heart disease responding to the usual chest survey could be detected. Thus, a special reading or a special view might draw attention to chest photofluorography as a case-finding device for heart disease when even with the best techniques only a small proportion of cases with existing disease could be identified. Additional information is also needed as regards the effect of using various criteria for interpreting films during the initial screening.

STUDY MATERIAL

Much clinical material is available which can be brought to bear on questions regarding the value of photofluorography as means of detecting heart disease. Appropriate are data arising from periodic physical examinations of the type provided by the Life Extension Examiners in New York City or by the Greenbrier Clinic at White Sulphur Springs, West Virginia. There appears to be little self-selection for these examinations so that inferences can prob-

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ably be made from these data to important and identifiable segments of our apparently healthy working population.

In this study, material from the Greenbrier Clinic was used. Some of the activities of the clinic have already been reported.[1] It was started in 1949 to provide complete periodic physical examinations for business executives and others and, at present, executives of some 50 companies receive periodic physical examinations. In addition, wives and children of these executives are sometimes examined. Many persons are now receiving their fourth or fifth examination. Thus, an excellent picture of the development of cardiovascular disease, as well as its prevalence, is available from the clinic records. Such data could be put to many uses and conceivably might provide some useful leads as to precipitating factors in the development of cardiovascular disease. In this study, only a limited amount of the material available has been drawn upon.

Posteroanterior and lateral 14 by 17 inch roentgenograms on 2,174 persons who had received a complete diagnostic examination at the Greenbrier Clinic were copied on 70 mm. roll film. The original films were not used because of their inaccessibility for study. Essentially, the films copied represented the first examination made by the clinic on all persons seen through June, 1954. Shown in Table 1 is the age and sex distribution of the population used, along with the number and percentage in each age and sex group who were diagnosed by the clinic as having heart disease. As might be expected, the prevalence of heart disease was higher in the older age groups and slightly higher among the males. Criteria of the New York Heart Association were rigidly followed in the establishment of these diagnoses. Examinations of patients consisted of a complete history and physical examination, posteroanterior and lateral chest roentgenograms (14 by 17 inch), electrocardiogram (12 leads) with double Master exercise test, and generally accepted laboratory studies. All of these were performed routinely; other diagnostic aids were employed whenever indicated. Records have been subsequently reviewed to doubly insure that diagnostic criteria were applied uniformly to the series of patients represented by this study. Among the 218 persons diagnosed as having heart disease or possible heart disease, there were 71 cases of arteriosclerotic heart disease, 69 cases of hypertensive heart disease, 21 cases of rheumatic heart disease, 42 cases of other heart disease (including congenital heart disease and pericarditis) or heart disease with etiology unknown, and 37

TABLE I

PERSONS EXAMINED AND HEART DISEASE DIAGNOSED BY AGE AND SEX SHOWING NUMBERS AND RATES

Age	Both Sexes			Male			Female		
	Number Examined	Diagnosed Heart Disease*	Per Cent Heart Disease	Number Examined	Diagnosed Heart Disease*	Per Cent Heart Disease	Number Examined	Diagnosed Heart Disease*	Per Cent Heart Disease
All ages	2,174	218	10.0	1,585	172	10.9	589	46	7.8
Under 30	45	1	2.2	21	1	4.8	24	0	0.0
30–39	329	14	4.0	218	7	3.2	111	6	5.4
40–49	719	40	5.6	533	32	6.0	186	8	4.3
50–59	777	100	12.9	569	76	13.4	208	24	11.5
60 and over	296	62	20.9	240	54	22.5	56	8	14.3
Age unknown	8	2	25.0	4	2	50.0	4	0	0.0

* Including possible heart disease.

cases of possible heart disease. A total of 240 cases of heart disease were diagnosed in 218 persons. Thus, there was a concomitance of diseases in some instances. Fifteen of the persons diagnosed with arteriosclerotic heart disease also had hypertensive heart disease. Two of the persons with rheumatic heart disease also had arteriosclerotic heart disease; 2 of the persons with arteriosclerotic heart disease were also considered as having other possible heart disease; and 3 persons with hypertensive heart disease also had other heart disease of unknown etiology.

As was stated previously, there appears to be little self-selection for examination at the Greenbrier Clinic, and the persons examined appear to be fairly representative of the group from which they were drawn (*i.e.*, largely business executives). Some evidence is available which suggests that, in terms of the prevalence of heart disease this group may also adequately describe larger working populations. In a study of Los Angeles Civil Service workers, a 10 per cent random sample of Los Angeles Civil Service employees was given a complete examination for cardiovascular disease.[9] It is probably of significance that the prevalence of heart disease found in the population examined at the Greenbrier Clinic is similar, particularly among males, to the prevalence reported in the Los Angeles study. A comparison of the prevalence of definite heart disease in the two groups is shown in Table II. While there were some differences in the etiologic distribution of heart disease between the Los Angeles and Greenbrier series, much of this can be explained on the basis of a difference in the age distribution of the groups involved. This suggests that the results of validation of chest roentgenography using the population screened at the Greenbrier Clinic is generally applicable to other important working groups.

TABLE II

PREVALENCE OF DEFINITE HEART DISEASE, EXAMINATIONS AT GREENBRIER CLINIC AND LOS ANGELES CIVIL SERVICE EMPLOYEES BY AGE AND SEX

Age	Male		Female	
	Greenbrier	Los Angeles	Greenbrier	Los Angeles
30–39	1.8%	3.7%	3.6%	2.2%
40–49	4.7	5.4	4.3	.9
50–59	11.4	11.5	9.1	14.5
60 and over	19.2	20.8	12.5	16.7

RESULTS

The posteroanterior chest roentgenograms available of the total 2,174 persons examined for heart disease were carefully read by one of us (AEB) without knowledge of the diagnosis established and, in fact, without any record except the 70 mm. film copy. All cardiovascular abnormalities were noted without regard to their probable clinical significance. Roentgenograms were classified as shown in Table III—with priority given in the order in which major classifications are shown in this table. Thus, where chest roentgenograms showed both enlargement and calcification of the aorta, they are included only in the enlargement categories; roentgenograms showing abnormalities in the shape of the aorta and calcification of the aorta are included only in the calcified aorta category, etc. Abnormalities in configuration without enlargement include straightening of the left cardiac border and prominent pulmonary conus. Isolated dextrocardia and complete situs inversus were not considered abnormal unless some other finding was present.

The ability of a screening test to identify persons with a particular disease is referred to as the sensitivity to the test. The sensitivity of chest roentgenography in the detection of heart disease can be readily estimated from Column 4 of Table III where various criteria are shown which might be used as the basis for referring persons for further study. One hundred and sixteen or 53.2 per cent of the 218 persons diagnosed as having definite or possible heart disease had some cardio-

TABLE III

READINGS ON POSTEROANTERIOR ROENTGENOGRAMS COMPARED WITH DIAGNOSIS MADE AT CLINIC

Roentgen Findings	Diagnosis Made at Clinic					
	Total		Heart Disease*		No Heart Disease	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
	(1)	(2)	(3)	(4)	(5)	(6)
Total read	2,174	100.0	218	100.0	1,956	100.0
No cardiovascular abnormality	1,645	75.7	102	46.8	1,543	78.9
Cardiovascular abnormalities	529	24.3	116	53.2	413	21.1
10–20% enlargement	108	5.0	24	11.0	84	4.3
20–40% enlargement	87	4.0	30	13.8	57	2.9
40% enlargement and over	28	1.3	17	7.8	11	.6
Slight abnormal configuration	30	1.4	4	1.8	26	1.3
Moderate or marked abnormal configuration	13	0.6	4	1.8	9	0.5
Calcification of aorta	114	5.2	18	8.3	96	4.9
Shape of aorta	146	6.7	18	8.3	128	6.5
Great vessels	1	.05	0	0.0	1	0.05

* Including possible heart disease.

vascular abnormality noted when their posteroanterior chest roentgenogram was read for this study.

Obviously, the more inclusive criteria for recall, the larger is the proportion of existing heart disease that can be detected; however, the disadvantages in making criteria for follow-up too inclusive are readily apparent from the data presented in Column 6 of Table III. This shows the ability of chest roentgenograms to properly identify persons without heart disease—the specificity of a screening test. It will be noted that only 78.9 per cent of the persons without heart disease had completely normal chest roentgenograms insofar as cardiovascular abnormalities were concerned. Of the 21.1 percent who had some abnormality, the bulk (11.4 per cent) were confined to abnormalities of the aorta, and it would appear that in general this is a poor criterion for routine follow-up. If criteria for follow-up were confined to heart enlargement or moderate or marked abnormalities in configuration, specificity is improved somewhat. Under these conditions, 8.3 per cent of the persons without heart disease would be recalled unnecessarily—or, put another way, 91.7 per cent of all persons without heart disease would be so identified (specificity)—while 34.4 percent of all persons with heart disease would be so identified (sensitivity). With these criteria chest roentgenography was most sensitive in detecting hypertensive and rheumatic heart disease. This might be expected and has been reported in other studies.[7,13]

Data presented in Table III are strictly applicable to screening chest surveys only insofar as the populations examined are similar to that used in this study. It is quite probable that there would be differences in the age and sex distribution. As can be seen on Table I, the persons in this study were predominantly male and tended to be older than the general population. Using heart enlargement plus moderate or marked abnormalities in configuration as the sole criteria for positivity, the sensitivity and specificity of posteroanterior roentgen films of the chest in detecting heart disease in various age and sex groupings is shown in Table IV. In general, chest roentgenography appears to be more sensitive for heart disease in females than in males. With

TABLE IV

SENSITIVITY AND SPECIFICITY OF CHEST ROENTGENOGRAPHY FOR HEART DISEASE* USING HEART ENLARGEMENT OR MODERATE OR MARKED ABNORMALITIES IN CONFIGURATIONS CRITERIA FOR POSITIVITY IN VARIOUS AGE AND SEX GROUPINGS

	Sensitivity	Specificity
	Per Cent of Persons with Heart Disease Screening Positive	Per Cent of Persons without Heart Disease Screening Negative
Males		
Under 50	22.5	92.9
50 and over	38.4	87.8
Females		
Under 50	35.7	95.1
50 and over	34.4	87.9

* Including possible heart disease.

regard to age, the sensitivity of chest roentgenography does not show any consistent pattern; however, the specificity is somewhat better in the younger age group than in the older. These data suggest that in the usual chest survey, where the population examined is generally somewhat younger than that used here and more evenly distributed as to male and female, if films were carefully examined for enlargement or moderate or marked abnormalities in configuration, the over-all sensitivity and specificity rates would be higher than those reported in this study.

In order to determine whether lateral chest roentgenograms in addition to posteroanterior views, would materially change the usefulness of chest roentgenograms in sorting out persons with heart disease, posteroanterior and lateral films were read in combination but under the same conditions as before. Comparing these readings with those shown in Table III for posteroanterior films alone revealed no significant differences, and it was concluded that in case-finding surveys where heart shadows are evaluated on the basis of the appearance of size or shape (as in this study), the addition of a lateral film would contribute little to their value in sorting out heart disease.

SIGNIFICANCE OF FALSE POSITIVES

It will be noted in Table III that many persons had cardiovascular abnormalities noted on their chest roentgenograms but were not diagnosed as having heart disease. These are usually referred to as "false positives." It is of interest to compare the frequency with which heart disease was subsequently diagnosed in this group with the frequency with which it was subsequently diagnosed in the "true negative" group, *i.e.*, persons whose chest roentgenograms were considered negative in this study and who were not diagnosed as having heart disease at the time of the initial roentgenography. Of the 1,956 persons in this study without heart disease at the initial examination, 762 had at least one subsequent examination at the Greenbrier Clinic. Actually, the percentage return is considerably higher (and more representative) than this would suggest since the numbers of examinations each year have increased steadily since 1949 when the Clinic was first opened. Thus, many of the persons in this study have not yet had an opportunity for re-examination. Of the 762 persons having a subsequent examination, 169 had some cardiovascular abnormality noted on chest roentgenography in this study (were "false positives") while 593 had a normal film (were "true negatives"). Of those with a normal film, 11 or 1.9 per cent were diagnosed as having heart disease or possible heart disease on a subsequent examination while of those with a positive film, 9 or 5.3 per cent were so diagnosed. While the numbers are small, it seems likely that "false positives" do develop heart disease at a greater rate than do "true negatives." Further studies of this type might ultimately lead to improvements in diagnostic criteria.

DISCUSSION

This study was undertaken for the purpose of making inferences as to the value

of chest photofluorographic surveys in the detection of heart disease. As practiced in routine chest surveys, the film readings validated were not a part of the clinical diagnosis, and thus the findings reported here reflect the differences of opinion between the physician interpreting the screening film and the physician making the diagnosis which would be observed on case-finding surveys. In some respects the true situation of photofluorographic surveys was not simulated. The chest roentgenograms studied were actually 14 by 17 films and, while they were reproduced to about the same size as a 70 mm. photofluorogram, they were taken at about half the exposure time as a photofluorogram, with the result that the heart motion was more nearly stopped than would have been the case had a photofluorogram been made. Moreover, they were taken at a 72-inch distance rather than at the 38-inch distance usually used for photofluorograms; thus, there was less distortion of the vascular shadow than is present in screening survey films. It is unlikely that these factors would seriously affect the applicability of the results presented, since with proper training there is probably little difference between the reading of 14 by 17 roentgenograms and photofluorograms in identifying abnormal cardiovascular shadows.[4,14]

The reproducibility of the film readings reported here is not known. One other reading of the chest roentgenograms used is available for comparison with those reported here, however. This is the reading which was made at the time of diagnosis by the radiologist at the Greenbrier Clinic. A comparison of these two sets of readings shows good agreement and it seems likely that the readings reported here are fairly reproducible.

The sensitivity and specificity of chest roentgenograms in sorting out heart disease where various film reading criteria are used are summarized in Table v. This table is based on material presented above except for data for tuberculosis readers which is based on a report by Phillips, *et al.*,[9] as well as unpublished data in the files of the Tuberculosis Program of the Public Health Service and other data. From this, comparisons between chest roentgenography and other screening devices proposed for heart disease can be made. Dawber, *et al.*,[2] report

TABLE V

SENSITIVITY AND SPECIFICITY OF CHEST ROENTGENOGRAPHY IN THE DETECTION OF HEART DISEASE* WHERE VARIOUS CRITERIA FOR FOLLOW-UP ARE USED

Film Reading Criteria	Sensitivity: Per Cent of Persons with Heart Disease Screening Positive	Specificity: Per Cent of Persons without Heart Disease Screening Negative
Tuberculosis reader (usual criteria)	4.3	99.9
Special reader		
(1) hearts enlarged 40% or more	7.8	99.4
(2) (1) plus hearts enlarged 20–40%	21.6	96.5
(3) (1)+(2)+moderate or marked abnormalities in configuration of heart	23.4	96.1
(4) (1)+(2)+(3)+hearts enlarged 10–20%	34.4	91.7
(5) (1)+(2)+(3)+(4)+calcification	42.7	86.9
(6) any abnormality	53.2	78.9

* Including possible heart disease.

that among 2,000 volunteers in a study at Framingham, Massachusetts, the Lead I electrocardiogram was abnormal in 30 per cent of the persons with heart disease and normal (or doubtful) in 90 per cent of the persons without heart disease. This compares quite favorably with chest roentgenography where criteria for follow-up are heart enlargement, 10 per cent or more, and moderate or marked abnormalities in heart configuration (sensitivity 34.4 per cent, specificity 91.7 per cent). Phillips, *et al.*,[9] report that in their study a history form with properly selected questions was positive in 50 per cent of the heart disease cases and negative in 82 per cent of the persons without heart disease. This might be compared with chest roentgenography where any abnormality was considered as evidence for follow-up (sensitivity 53.2 per cent, specificity 78.9 per cent). Thus, with proper selection of reading criteria, chest roentgenography seems to compare favorably with other screening devices proposed for heart disease.

SUMMARY

1. A study was conducted to determine the proportion of the heart disease existing in our population which photofluorographic chest surveys are capable of detecting, to examine various criteria for reading survey films, and to determine the value of a lateral view in addition to the usual postero-anterior view for heart disease screening.

2. Material used was from the Greenbrier Clinic in White Sulphur Springs, West Virginia, where periodic physical examinations are made. As far as could be determined, this group seemed to be generally representative of other working groups as regards heart disease, and the results of this study would seem to estimate the usefulness of chest photofluorographic screening surveys conducted as a tuberculosis case-finding measure.

3. If all abnormalities of the heart and great vessels seen on the films were considered as indication for recall for follow-up, it is estimated that chest photofluorograms could detect 53 per cent of the heart disease existing in our population. This would result in 21 per cent of the persons without diagnosable heart disease being recalled. If enlargement of 20 per cent or more, or moderate or marked abnormalities in configuration were used as criteria for recall, 23 per cent of the heart disease cases could be identified and only 4 per cent of the persons without diagnosable heart disease would be recalled.

4. The addition of a lateral view of the chest did not improve the ability of the film reader in sorting out heart disease.

5. False positives were diagnosed as having heart disease on subsequent roentgen examination more frequently than were true negatives.

6. With proper selection of reading criteria, chest photofluorography compares favorably with other screening devices for heart disease.

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ROENTGEN MANIFESTATIONS OF PULMONARY ARTERIOVENOUS FISTULA*

DIAGNOSIS AND TREATMENT OF FOUR NEW CASES

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PULMONARY arteriovenous fistulas are congenital hemangiomatous malformations of the pulmonary vascular bed. Their diagnosis depends upon roentgenography. Over 170 cases have been described since the first clinical report was made in 1939.[10,14,17,19,21,23,24] There is general agreement that the best treatment for pulmonary arteriovenous fistulas is surgical excision of the lesion, with removal of as little lung as possible, in order to preserve lung function, especially if multiple fistulas are present. Surgery is curative and symptoms such as dyspnea, cyanosis, polycythemia and clubbing of digits dramatically disappear. Excision of fistulas is also advocated as a prophylactic measure in asymptomatic patients; the risk of segmental resection or lobectomy is far less than the morbidity and mortality of complications such as brain abscess, cerebral vascular accidents or hemoptysis.[3,21]

Since 1950, 13 patients with pulmonary arteriovenous fistulas have been studied at this Center; 9 have previously been reported;[21] 4 new cases are herein recorded. The roentgen findings of abnormal pulmonary shadows are the most constant features of the disease; angiocardiography especially provides the definitive diagnosis. The purpose of this paper is to summarize the roentgen aspects of pulmonary arteriovenous fistulas, to emphasize that the diagnosis is often the reward of a high index of suspicion, and to report the diagnosis and treatment of 4 new cases.

REPORT OF CASES

CASE 1. *Multiple Pulmonary Arteriovenous Fistulas, Left Lung, Associated with Hemoptysis, Cyanosis, Polycythemia, Clubbing of Digits and Syncope: Cure by Lobectomy.* A fifty-nine year old engineer (N.Y.H. No. 706554) was referred by Dr. George C. Adie on March 29, 1955. Eighteen months earlier the patient had had a sudden hemoptysis and for the first time his attention was called to cyanosis of the lips and clubbing of the digits. Six months afterwards, chest roentgenograms disclosed pulmonary densities in the left lung which were suspected of being due to neoplasm. Bronchoscopy early in January, 1954, disclosed a "polyp" of the left main bronchus; smears of the bronchial washings failed to show neoplastic cells. An exploratory left thoracotomy on January 8, 1954, revealed a 3 cm. mass in the lower portion on the left upper lobe which was excised. Pathologically, it was reported as "hamartoma (hemangioma)." After the operation the patient was well. On March 8, 1955 he had a sudden "blackout," and was rushed to a hospital. Subsequently, because of dyspnea, cyanosis, clubbing of the digits and the biopsy report of a hemangioma, pulmonary arteriovenous fistulas were suspected. He was referred to The New York Hospital for angiocardiography.

On examination, there was cyanosis of the lips and finger nails and the digits were markedly clubbed. Examination of the chest revealed a well healed left thoracotomy scar. The left lung was dull, the breath sounds were diminished; no rales were present. A bruit was not heard. The heart was not enlarged; the rate was regular and there were no murmurs; the blood pressure was 130/70. Laboratory data showed: hemoglobin, 18 gm./100 cc.; erythrocytes, 5.9 m./cu. mm. Roentgenography of the chest (Fig. 1*A*) showed a cluster of rounded densities in the left mid-lung field just outside the hilus and in the left lower lobe. Angiocardiography on March 29, 1955, revealed pulmonary arteriovenous fistulas in the lingular segment of the left upper lobe and basilar segments of the left lower lobe (Fig. 1*B*).

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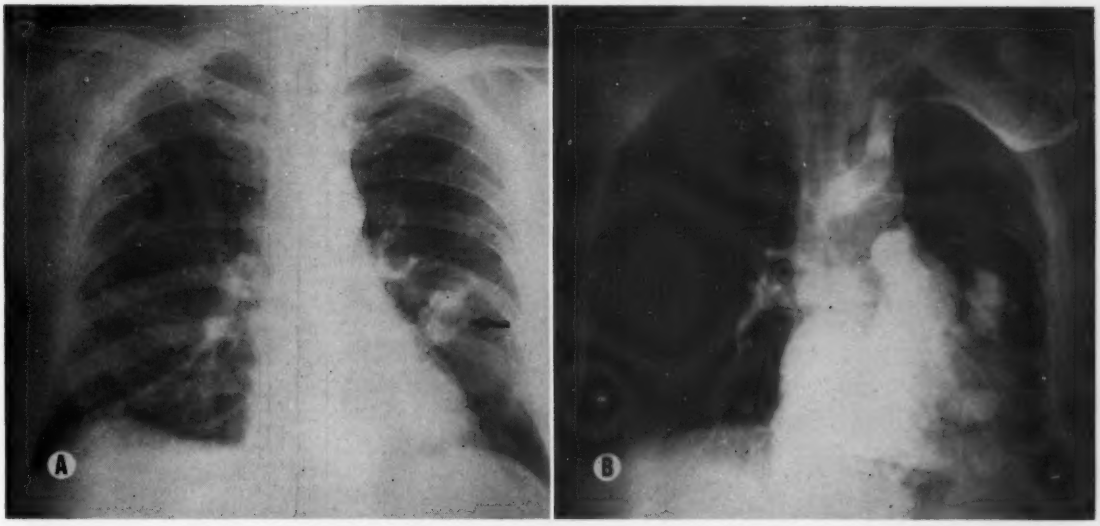

Fig. 1. Case I. *Multiple pulmonary arteriovenous fistulas in left lung discovered after hemoptysis and syncope, and associated with exertional dyspnea, cyanosis, polycythemia and clubbing of the digits. Cure by lobectomy.* (*A*) Conventional frontal roentgenogram of a fifty year old man shows a cluster of rounded densities in lower left hilus (arrow). Other masses at left base are hidden by the heart. (*B*) Frontal angiocardiogram reveals pulmonary arteriovenous fistulas near left hilus, located in the lingular segment of the left upper lobe (lateral films not shown). Note also the multiple pulmonary arteriovenous fistulas in several basilar segments of the left lower lobe.

On May 4, 1955, Dr. George C. Adie performed a left pneumonectomy. The patient had an uncomplicated postoperative recovery. In due time, he was able to resume his regular employment. Now, dyspnea occurs only after overexertion; cyanosis and clubbing of the digits have completely disappeared.

Pathologic study of the excised left lung disclosed a 2.5 by 1.8 cm. pulmonary arteriovenous fistula of the left upper lobe and multiple arteriovenous fistulas of the left lower lobe. Histologic examination of many sections showed marked intimal sclerosis of the arterial components of the fistula; in contrast, the venous channels had only slight intimal sclerosis. No inflammatory cells were seen within the lung. Review of the earlier biopsy specimen of the left lung taken on Jan. 8, 1954, disclosed the presence of an artery entering a plexus of undifferentiated vessels. The diagnosis of hamartoma was revised and instead a lesion consistent with the presence of arteriovenous fistulas was assumed.*

Case II. *Pulmonary Arteriovenous Fistula, Lingular Segment, Left Upper Lobe, Associated with Rendu-Osler-Weber Disease and Hemiplegia: Lingulectomy.* A fifty year old housewife was referred by Drs. Frances Karp and Vincent J. Governale on July 25, 1955. The patient had had nose bleeds all her life and two years previously also had had bleeding of the tongue. Suddenly, on May 5, 1955, she became dizzy and fell to the floor. She was sent to a hospital, found to have right hemiplegia and slurred speech. Subsequently, she improved and recovered motion of the right extremities; the slurred speech, however, persisted. The patient's two children and brother have had nose bleeds; her mother died of a "brain tumor" at the age of fifty-nine years.

On physical examination, the patient was well developed and nourished and showed increased reflexes and weakness of the right arm and leg. A 2 by 2 cm. hemangioma was present at the tip of the tongue. No bleeding or telangiectasia was seen in the nares. There was no clubbing or cyanosis of the digits. The heart was not enlarged and there were no murmurs; the blood pressure was 110/74. A to-and-fro bruit, accentuated with deep inspiration and obliterated on expiration, was heard at the apex of the heart. The rest of the examination was normal. Frontal and lateral roentgenograms showed a globular density 1 by 2 cm. in diameter in the lingular segment of the left upper lobe (Fig. 2*A*). Characteristic afferent and effer-

* Courtesy of Dr. William C. Schraft, Jr.

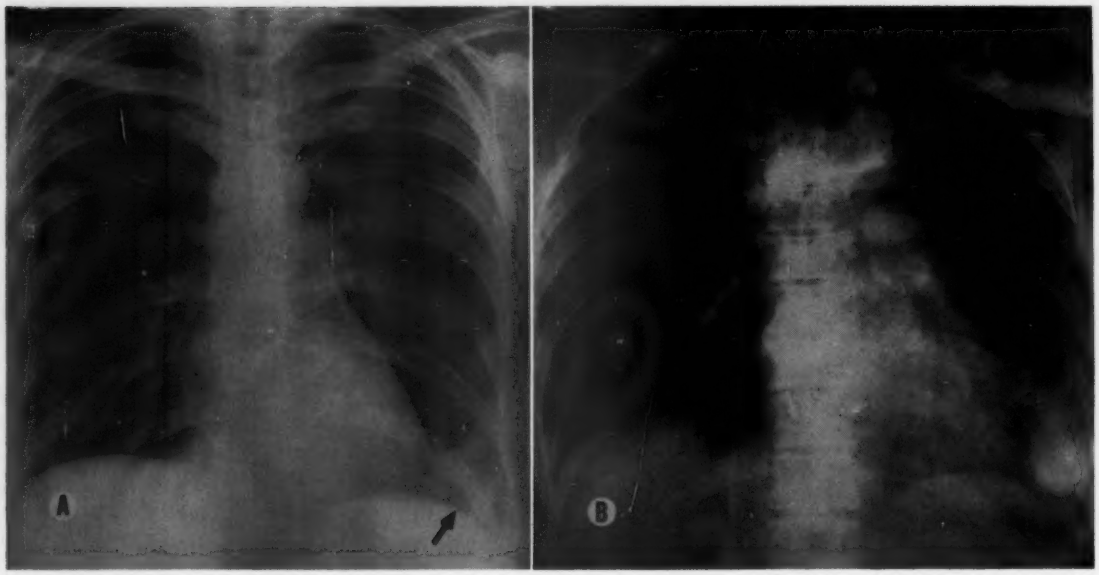

FIG. 2. Case II. *Pulmonary arteriovenous fistula of the lingular segment, left upper lobe; diagnosis made after right hemiplegia. Triad of Rendu-Osler-Weber disease (epistaxis, telangiectasia and familial tendency) present.* (*A*) Conventional roentgenogram of a fifty year old woman shows density of a hazy character left base (arrow) connected to hilus by afferent and efferent vessels. (*B*) Frontal angiocardiogram shows opacified arteriovenous fistula. (Courtesy of Dr. J. L. Mangiardi.)

ent vessels connecting the mass and left hilus were clearly seen.

Angiocardiography performed by Dr. Joseph L. Mangiardi (Fig. 2B) demonstrated the arteriovenous fistula. On August 10, 1955, excision of the lingular segment of the left upper lobe was performed by Dr. Mangiardi. There was immediate disappearance of the bruit and thrill; the postoperative course was uneventful and the patient was discharged on the tenth postoperative day.

CASE III. *Asymptomatic Pulmonary Arteriovenous Fistula, Medial Basal Segment, Left Lower Lobe: Segmental Resection.* A twenty-two year old sailor (N.Y.H. No. 729263) was referred for angiocardiography on Jan. 17, 1956. Routine chest roentgenography had disclosed a rounded 3 by 3 cm. density in the medial basal segment of the left lower lobe which was connected to the left hilus by vessels (Fig. 3, *A* and *B*).

On physical examination there was a systolic (grade 3) bruit at the base of the heart which was accentuated with inspiration; elsewhere there were no abnormal findings. There was no familial history of bleeding or telangiectasia. Laboratory data revealed: hemoglobin, 15.0 gm./100 cc; hematocrit, 50 per cent; arterial oxygen saturation was normal. Angiocardiography (Fig. 3, *C* and *D*) clearly demonstrated an arteriovenous fistula confined to the medial basal segment of the left lower lobe. On Jan. 19, 1956, the medial basal segment of the left lower lobe was excised and the patient made an uneventful recovery.

CASE IV. *Pulmonary Arteriovenous Fistula, Lateral Basal Segment, Right Lower Lobe, Associated with Rendu-Osler-Weber Disease: Right Lower Lobectomy.* A twenty-eight year old white housewife (N.Y.H. No. 725669) was admitted on Dec. 4, 1955 with a complaint of nose bleeds occurring daily all her life. These had come spontaneously often from both nostrils and in gushes requiring packing. At the age of fourteen years, profuse bleeding followed tonsillectomy requiring suture. The next year a routine chest roentgenogram in school disclosed a "spot" in the right lung and she was confined to a sanatorium for eight months. About that time, she first noted a "red spot" in the left upper cheek.

Two months prior to admission, during the fifth month of pregnancy, multiple "red spots" appeared on the thorax, arms and back; these disappeared after delivery. To her delight the nose bleeds completely subsided following delivery. Dyspnea, cyanosis and headaches have

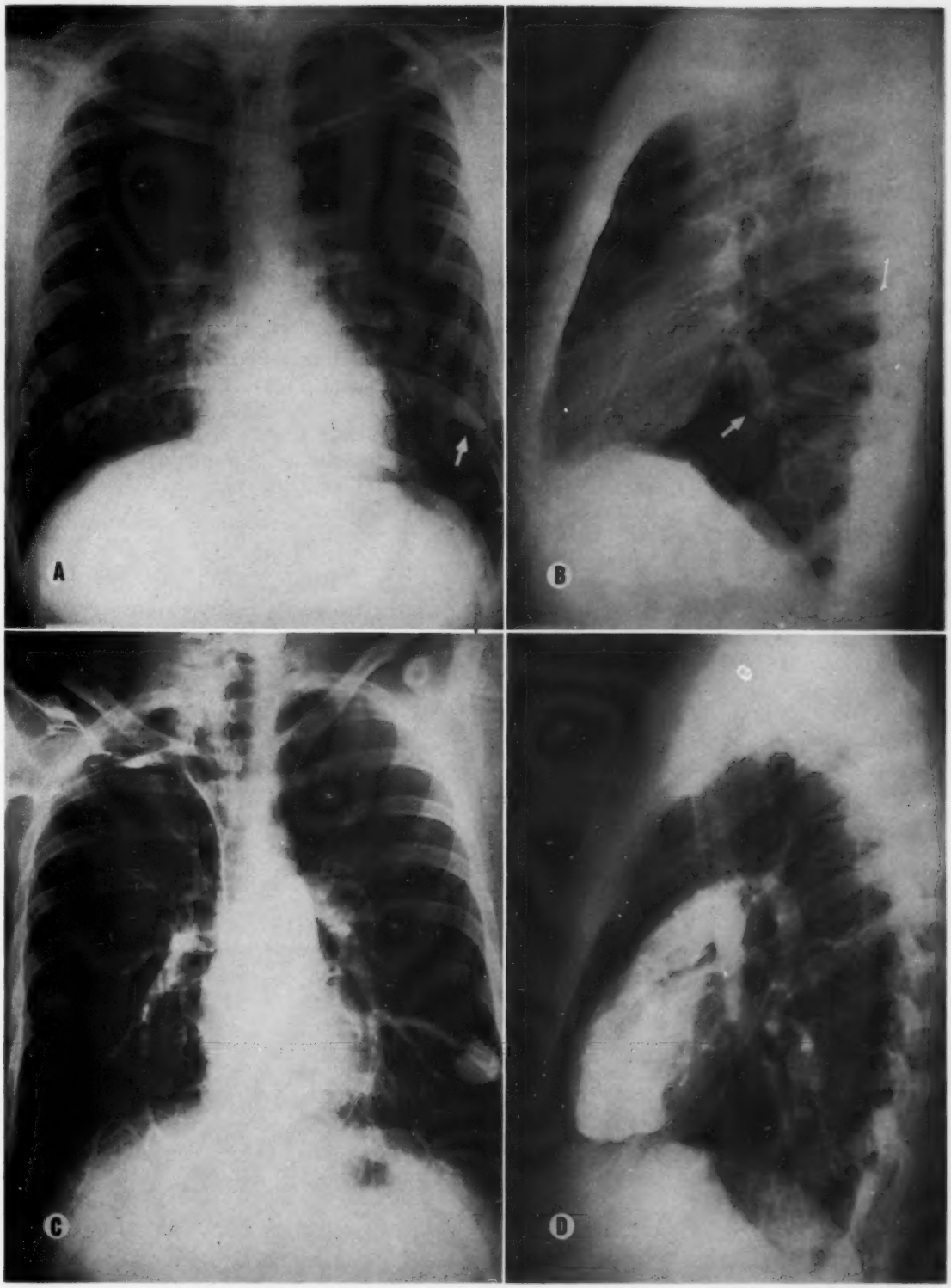

FIG. 3. Case III. *Asymptomatic pulmonary arteriovenous fistula of the medial basilar segment of the left lower lobe in a twenty-two year old man.* (*A*) Frontal chest roentgenogram shows a rounded mass in left lower lung field (arrow). (*B*) Lateral roentgenogram also shows the rounded lesion (arrow). (*C*) Frontal angiocardiogram clearly delineates the arteriovenous fistula. (*D*) Lateral angiocardiogram also shows and localizes the opacified arteriovenous fistula.

never been present and there was no family history of bleeding among four sisters, a brother and her parents.

On physical examination the patient was slightly pale, noncyanotic, well developed and nourished. There were multiple telangiectasias and a hemangioma of the lower lip (Fig. 4*A*). A large red spider telangiectatic lesion which blanched on pressure was present below the left eye; smaller ones were seen on the right cheek and beneath the finger and toe nails (Fig. 4*B*). No bleeding was recognized in the nostrils. There was no clubbing of the digits. An area of telangiectasis of the nasal septum was seen 2 cm. from the left vestibule. Petechial lesions were also present in the posterior pharangeal wall and tongue. The heart was not enlarged, the rate was regular and there were no murmurs; the blood pressure was 102/54. A soft to-and-fro vascular bruit, increased in intensity with inspiration, was heard in the eighth interspace, low in the right midaxilla. The rest of the examination was normal.

Laboratory data were as follows: hemoglobin, 14.0 gm./100 cc.; hematocrit, 44 per cent; leukocytes, 7,400 with a normal differential; bleeding time 1.2 minutes; and clotting time, 5.3 minutes. The electrocardiogram was normal. The chest roentgenograms (Fig. 4, *C* and *D*) showed a cluster of small rounded densities (1.5 to 2 cm. in diameter) intimately related to the lung vasculature and located in the lateral basal segment of the right lower lobe. Angiocardiography (Fig. 4, *E* and *F*) clearly revealed the afferent pulmonary artery, the opacified fistula and efferent pulmonary veins proceeding toward the left atrium. At operation on January 23, 1956, three 1½ cm. sacs were located in the lateral basal segment of the right lower lobe. These were connected by a large pulsatile pulmonary arterial branch and multiple pulmonary venous channels. Lobectomy was done because of the widespread efferent pulmonary venous channels. A low melting point alloy cast of the specimen (Fig. 4*G*) was made by Dr. F. Stephen Vogel. It revealed a large pulmonary arterial communication connecting to clusters of dilated thin-walled 1.5 to 2.0 cm. sacs drained by a huge pulmonary vein which was connected to the pulmonary venous circulation of the entire lower lobe. Recovery from the operation was uneventful and the patient was discharged after eight days.

DISCUSSION

In recent years, many extensive articles on pulmonary arteriovenous fistula have appeared;[10,14,17,19,23,24] a review of the literature is, therefore, unnecessary. It suffices merely to point out that the 4 patients herein reported are representative examples of the disease. Case III, for instance, was asymptomatic and found after a routine roentgen survey of the chest. In contrast, Case I had onset with hemoptysis and syncope; Case II was discovered after hemi-

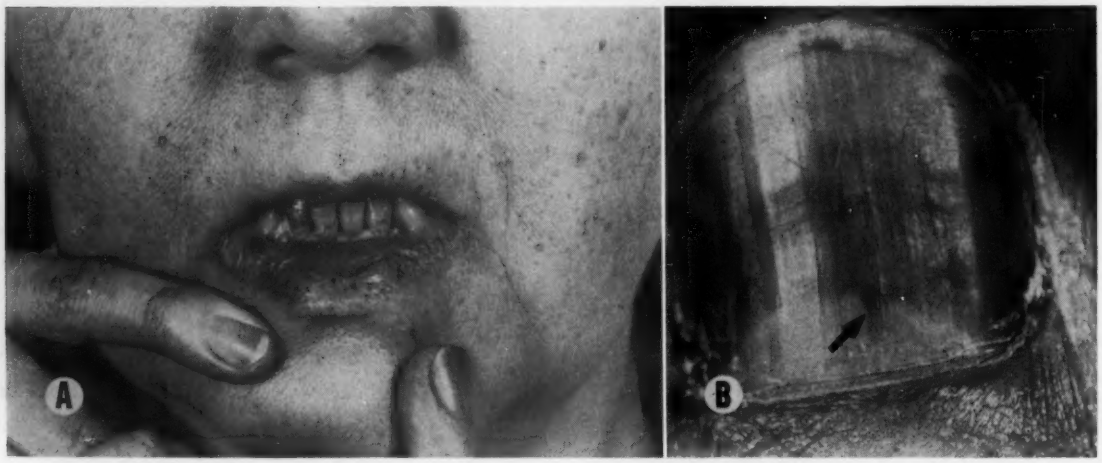

FIG. 4. Case IV. *Rendu-Osler-Weber disease associated with pulmonary arteriovenous fistula of the medial basal segment, right lower lobe.* (*A*) Photograph of a twenty-eight year old woman showing a hemangioma and capillary telangiectasia of the lower lip. (*B*) Photograph of capillary telangiectasia of nail bed (arrow).

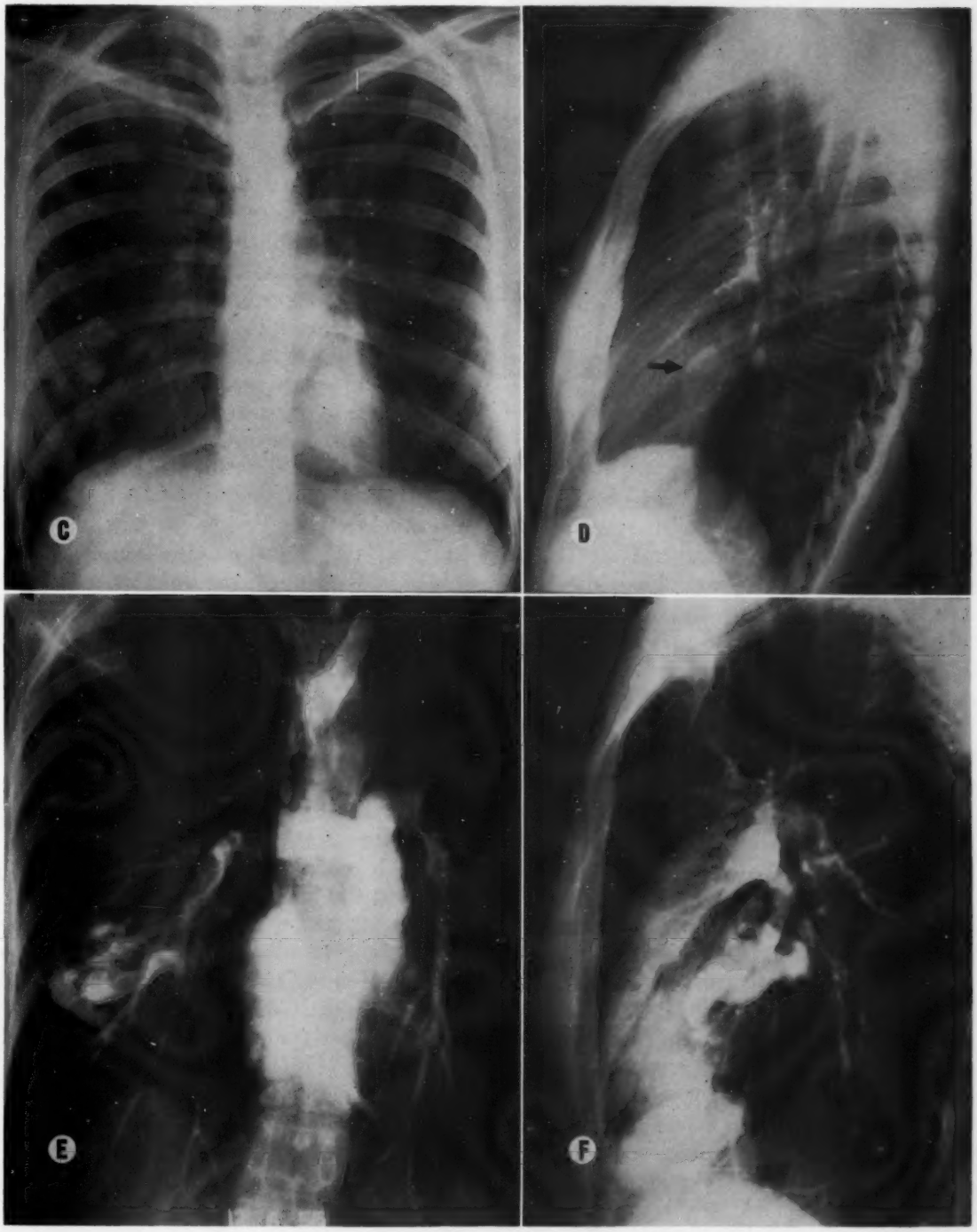

Fig. 4. Case iv. (*C*) Frontal chest roentgenogram shows a cluster of rounded densities in lower right lung field. (*D*) Lateral roentgenogram locates the lesions in the lateral basal segment of the right lower lobe (arrow). (*E*) Frontal angiocardiogram clearly reveals the cluster of pulmonary arteriovenous fistulas with the afferent (arterial) and efferent (venous) channels. (*F*) Lateral angiocardiogram also shows the fistulas.

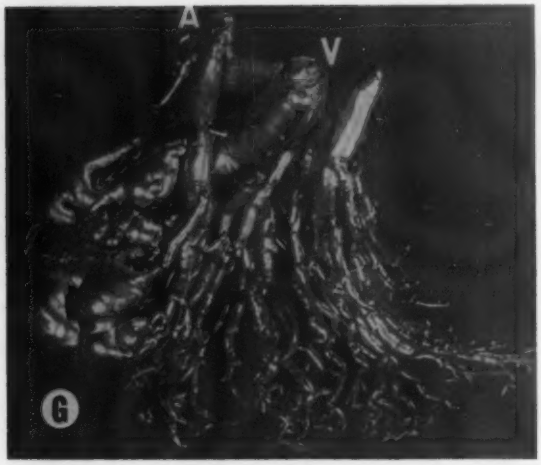

Fig. 4. Case IV. (*G*) Low melting point alloy cast of the fistula. A, is the afferent artery; V, is the efferent vein (Courtesy of Dr. F. Stephen Vogel.)

plegia; while Case IV had a life-time history of epistaxis. Only 1 patient (Case I) had the classic form with exertional dyspnea, cyanosis, polycythemia and clubbing of the digits, and this occurred probably because of the presence of multiple fistulas of the left lung. In contrast, the 3 other patients were acyanotic and had segmental lesions. Two of the patients (Cases II and IV) also had Rendu-Osler-Weber disease, indicating the close relationship of the two conditions.[21]

The complexity and multiplicity of the vascular connections of pulmonary arteriovenous fistulas may not be readily apparent after clinical and roentgenographic study. Low melting point alloy casts of the excised lesions[9] are important for visualizing the ramifications of the vessels.[21] For instance, the cast of the excised pulmonary arteriovenous fistula (Fig. 4*G*) shows that the pulmonary venous blood supply of the entire right lower lobe was intimately related to the fistula and explains why lobectomy rather than segmental resection was necessary to cure the condition. The rich admixture with pulmonary venous blood in the pulmonary arteriovenous fistula also explains the absence of cyanosis in this patient. Physiologic studies may be expected to show the degree of unsaturation of the blood in such cases[4] but will not readily reveal the shunts associated with the fistula.

Summary of Clinical Findings. Since 1950, a total of 13 cases of pulmonary arteriovenous fistula have been studied at this Center. This is a select group of a rather uncommon disease; only 5 were encountered among patients in the clinic and hospital; 4 others were referred from other hospitals because of the suggestive roentgen findings and/or vascular bruits; and 4 had had angiocardiography but were sent for more complete angiography of the lungs. Eight patients were females; 5 were of the male sex; and all were Caucasians. The ages at the time of the diagnosis varied from nineteen to sixty-seven years; mean, thirty-eight years. The right lung was the site of the fistula in 7 patients; the left lung was also involved in 7 (1 patient had bilateral disease). The right middle lobe was involved in 1 instance; the lingular segments of the left upper lobe, 3 times; and the left lower lobe, twice. A segmental pulmonary arteriovenous fistula was found in the right upper lobe (superior segment) and in the left upper lobe (anterior segment). Basal segmental fistulas of the right lower lobe were present in 4 instances; and in the left lower lobe in 3 instances. These findings are in accord with reports in the literature that there is a tendency for the fistula to occur mostly in the lower lobes.

Of the 13 cases, 4 (30 per cent) had the stigmata of familial hemorrhagic telangiectasia (Rendu-Osler-Weber disease); *i.e.*, epistaxis, anemia, capillary telangiectasis of skin, nail bed (Fig. 4*B*) and/or mucous membranes (Fig. 4*A*); hemangioma (Fig. 4*A*); and a familial history of the disease.[6,18] Five patients (38 per cent) were asymptomatic; 2 of these were siblings,[3] another indication of the congenital nature of the malformation. In 8 patients the presenting symptoms were: epistaxis, 3 instances; hemoptysis, once; hemiplegia, twice (brain abscess and cerebral thrombosis respectively); and meningo-encephalitis, once. Dyspnea, cyanosis, polycythemia and clubbing occurred twice; in both patients they

were associated with extensive fistulas of the left lower lobe.

Vascular bruits over the site of the pulmonary arteriovenous fistulas were heard in all but two patients. The murmurs varied from a faint systolic sound accentuated by deep inspiration to a loud harsh systolic blow; in one instance the bruit was present during diastole as well. Accurate localization of the fistula by roentgenography in frontal and lateral views aided in eliciting the auscultatory signs. Excision of pulmonary arteriovenous fistula was curative; 1 patient had a pneumonectomy; 5 had lobectomy and 4 had segmental resections. One patient refused operation and 2 patients were not offered surgery because of complications.[21]

ROENTGEN FEATURES

The most constant finding in pulmonary arteriovenous fistulas is the presence of abnormal shadows in the conventional chest roentgenogram. The fistulas may vary in size and shape from minute circular densities to large oval lesions (Fig. 1*A*, 3, *A* and *B* and 4, *C* and *D*) Vague and ill defined edges may simulate pulmonary infiltrates (Fig. 2*A*, 3*B*, 4, *C* and *D*). At times, localized increase of the hilar pulmonary vasculature should suggest an arteriovenous fistula (Fig. 1*A*), especially when lateral or oblique views indicate that the increased vasculature is related to the segmental or lobar pulmonary blood supplies. Another feature, the afferent pulmonary arterial and efferent pulmonary venous channels connecting with a pulmonary lesion, may be evident (Fig. 2*A*). Rarely anomalous pulmonary venous drainage from the right lung into the inferior vena may simulate a pulmonary arteriovenous fistula.[15] Calcification in pulmonary arteriovenous fistula is uncommon and has been reported in only 3 instances.[17]

Roentgenoscopy permits recognition of vascular pulsation of pulmonary arteriovenous fistulas and their vascular connections, but in none of the cases was this phenomenon observed. More important are the changes in size of the lesions during respiratory maneuvers. With the Valsalva procedure (expiration against the closed glottis), the intrathoracic pressure is raised, blood flow diminishes in the thorax and the fistula becomes smaller. The Müller procedure (deep inspiration against the closed glottis) decreases the intrathoracic pressure and more blood fills the fistula and makes it larger.

Laminagraphy has been highly recommended for the diagnosis of pulmonary arteriovenous fistulas.[5,7] In some instances it may be expected to supplement conventional roentgenography and disclose vessels connecting with the fistula. However, disease processes like bronchiectasis and chronic pneumonia may be exaggerated and reveal patterns that simulate pulmonary arteriovenous fistulas.[21]

In the roentgenographic differential diagnosis, primary or secondary lung tumors, bronchiectasis and tuberculosis are some of the conditions to be considered.[14,17,22] One patient (Case 1) was suspected of having a neoplastic lung lesion because of the presence of clubbing of the digits and densities in the left lung. Another patient (Case IV) was suspected of having tuberculous infiltrates, while in Case 1 a hamartoma was suspected.

Angiocardiography. There is general agreement that angiocardiography provides the definitive diagnosis of pulmonary arteriovenous fistula. Many authors have called attention to the importance of making large (14 by 17 inch) roentgenograms in order to prevent overlooking multiple and bilateral pulmonary arteriovenous lesions. Apparently, minute pulmonary telangiectasis may not be readily discernible by angiocardiography.[1,17,24] However, arteriovenous fistulas in pulmonary segments are easily recognized (Fig. 1*B*, 2*B*, 3, *C* and *D* and 4, *E* and *F*). The afferent and efferent vascular connections of pulmonary arteriovenous fistulas may be difficult to visualize with conventional roentgenography but are readily seen with angiocardiography. Multiple serial films are sometimes desirable for

complete study of the lesion during the arterial and venous filling stages but usually two large films taken with a standard stereo-cassette changer[2,12] at 2½ and 6 seconds after the beginning of the injection will show the pulmonary arterial and venous components of the fistula. Involvement of the chest wall by pulmonary arteriovenous fistulas has been reported.[11] With angiocardiography this complication might have been recognized.

Figure 5 (through the courtesy of Dr. Harold A. Lyons[8]) clearly illustrates the value of angiocardiography in the diagnosis and treatment of pulmonary arteriovenous fistulas. Bilateral fistulas (of the middle lobe on the right, and of the anterior segment of the upper lobe on the left) are demonstrated (Fig. 5, *A* and *B*). Middle lobe lobectomy was done; Figure 5*C* is a photograph of the excised specimen. Subsequently, the segmental fistula of the left upper lobe was also excised. Figure 5*D* is the postoperative angiocardiogram and shows the normal pulmonary vascular tree after removal of the bilateral pulmonary arteriovenous fistulas.

Figure 6 illustrates how angiocardiography is useful in excluding the presence of a pulmonary arteriovenous fistula. Conventional frontal and lateral roentgenograms (Fig. 6, *A* and *B*) were interpreted as showing a pulmonary arteriovenous fistula of the posterior basal segment of the right lower lobe. A bruit was not elicited and roentgenoscopy did not exclude the presence of a pulmonary arteriovenous fistula. Angiocardiography (Fig. 6*C*), however, clearly demonstrated the nonvascular character of the lesion and at operation a bronchogenic carcinoma was found.

In the literature there are two reports of untoward reactions following angiography in patients with pulmonary arteriovenous fistulas. A fatality[16] occurred after the injection of 35 cc. of a contrast agent through a cardiac catheter with the tip in the right atrium. Another patient[13] recovered after severe hemoptysis which followed injection of contrast material through a cardiac catheter placed in the main pulmonary artery. The recent report of rupture of the heart by injection of the contrast substance through a cardiac catheter during selective angiography[20] also emphasizes the danger of injecting contrast substances directly into the cardiovascular system via catheters.

In marked contrast is our experience with the intravenous method of angiocardiography. In all, 3,010 injections of contrast material have been made in 2,239 patients during the period from October, 1946 to April, 1956. Only one death occurred, this in a fifty-six year old woman who had a pericardial effusion, probably secondary to irradiation for breast cancer. This record, in a series containing many patients seriously ill with heart and lung disease, fully justifies the use of angiocardiography, especially in pulmonary arteriovenous fistulas, for in this disease preoperative information in regard to bilateral or multiple involvement of the lungs is essential for the planning of the operation and conservation of lung tissue. In spite of recent warnings,[14] cardiac failure and/or hypertension are not contraindications to angiocardiography.

SUMMARY

Pulmonary arteriovenous fistulas are hereditary hemangiomatous malformations of the pulmonary vascular bed. Almost 40 per cent of the reported cases also have hemangiomas elsewhere in the body, indicating that a close relationship to familial telangiectasis (Rendu-Osler-Weber disease) exists.

When the pulmonary arteriovenous fistulas are large and do not have systemic arterial connections, dyspnea, cyanosis, clubbing of the digits and polycythemia result from unsaturation of the blood. Hemoptysis may occur when there is rupture of the paper-thin pulmonary vascular wall There is also a high incidence of cerebral symptoms due either to cerebral thrombosis or secondary to the polycythemia and infection, especially brain abscess. The

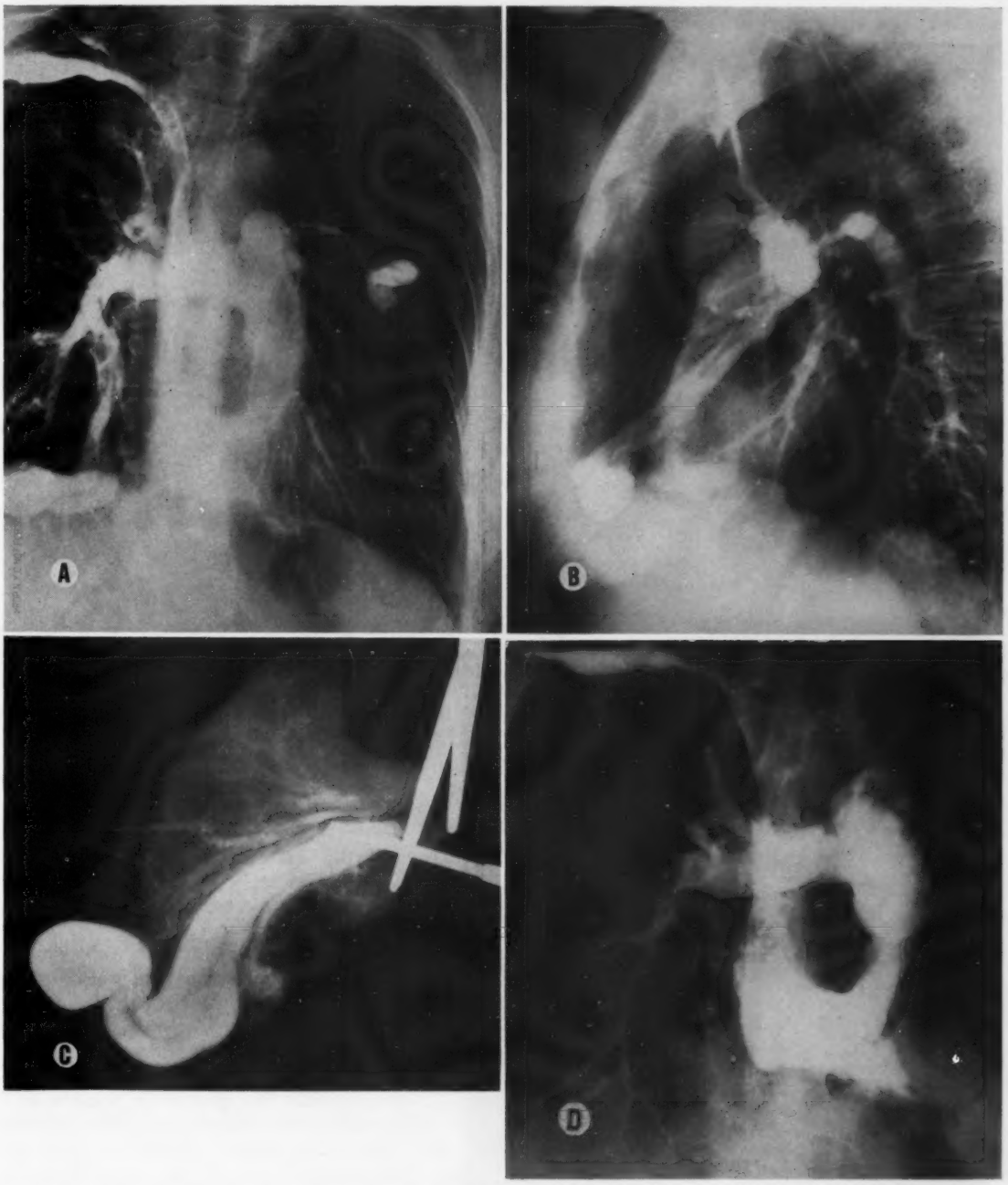

Fig. 5. *Bilateral pulmonary arteriovenous fistulas in a sixty-two year old woman with Rendu-Osler-Weber disease.* (Courtesy of Dr. H. A. Lyons and the *New England J. Med.*[8]) (*A*) Frontal angiocardiogram reveals a large pulmonary arteriovenous fistula just above the right diaphragm. Another is in the left mid-lung field. (*B*) Lateral angiocardiogram shows the right lung pulmonary arteriovenous fistula in the middle lobe. The vascular connections to and from the fistula are clearly identified. The pulmonary arteriovenous fistula of the left upper lobe was located in the anterior segment (arrow). (*C*) The excised middle lobe arteriovenous fistula injected with urokon. (*D*) Postoperative angiocardiogram shows a normal pulmonary arterial system.

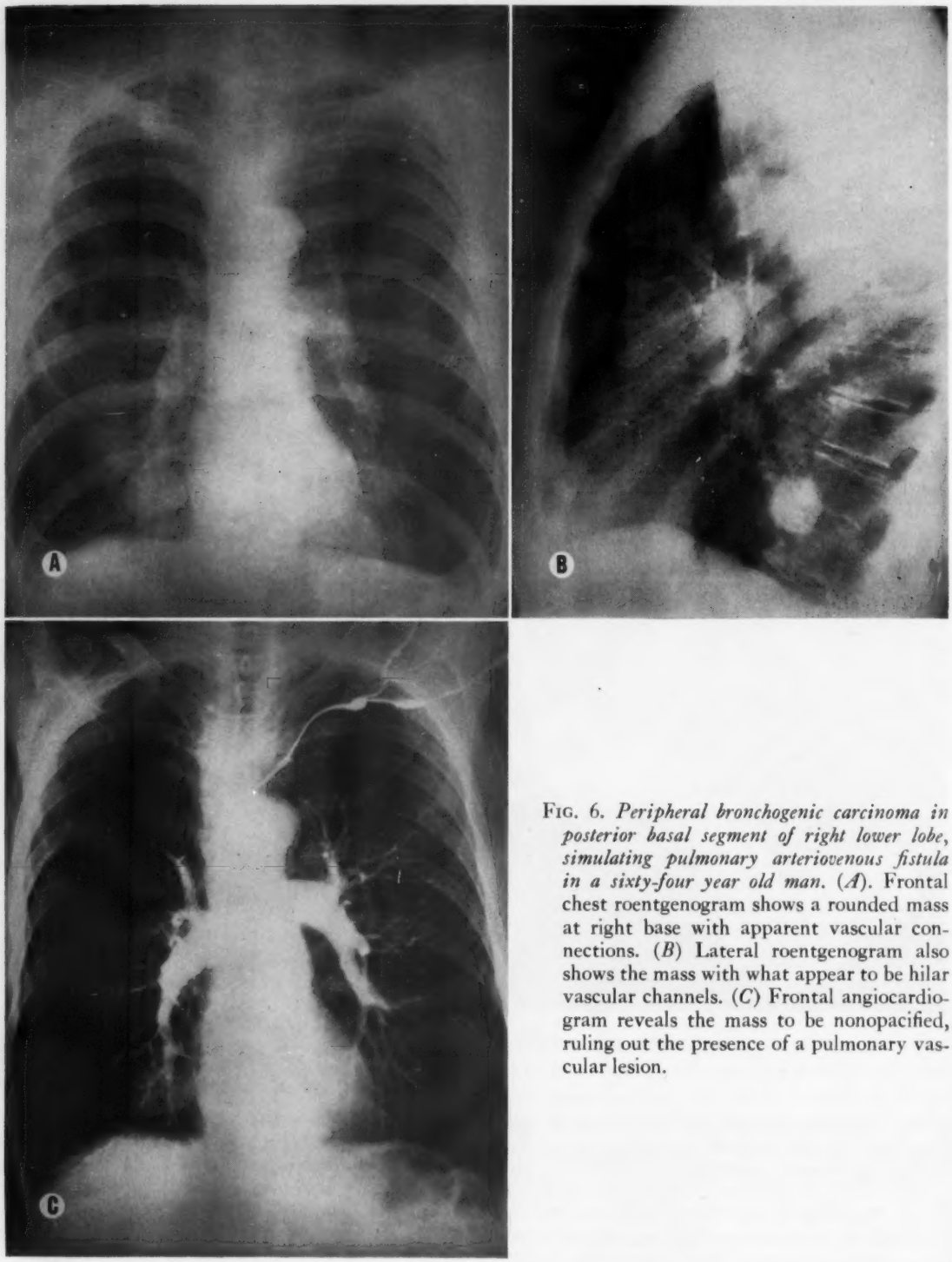

FIG. 6. *Peripheral bronchogenic carcinoma in posterior basal segment of right lower lobe, simulating pulmonary arteriovenous fistula in a sixty-four year old man.* (*A*). Frontal chest roentgenogram shows a rounded mass at right base with apparent vascular connections. (*B*) Lateral roentgenogram also shows the mass with what appear to be hilar vascular channels. (*C*) Frontal angiocardiogram reveals the mass to be nonopacified, ruling out the presence of a pulmonary vascular lesion.

heart remains normal in size because pulmonary arteriovenous fistulas do not cause increased pulmonary vascular resistance.

In our series of 13 cases the classic syndrome of cyanosis, clubbing of digits, polycythemia and a vascular murmur heard over the pulmonary fistula was present only twice. One patient with dyspnea, fatigue and familial hemorrhagic telangiectasis had bilateral pulmonary arteriovenous fistulas. In 5 instances (38 per cent) the patients were asymptomatic, in 8 the pulmonary arteriovenous fistula was responsible for symptoms, and in 3 of these, acute cerebral conditions (brain abscess, meningoencephalitis and hemiplegia) were present. Vascular bruits were heard in 11 patients; all had abnormal chest roentgenograms.

The diagnosis of pulmonary arteriovenous fistula can often be made by conventional roentgenography, particularly if the index of suspicion is high. Pulmonary densities with hilar vascular connections localized in lobes or segments of the lungs, and increased hilar vascularities, should arouse suspicion of an arteriovenous fistula. Change in size of the lesions after respiratory maneuvers (Valsalva, Müller) and delineation of the afferent arterial and efferent venous connections by laminagraphy may be confirmatory. Angiocardiography conclusively establishes the diagnosis and is especially recommended for preoperative evaluation.

The treatment of pulmonary arteriovenous fistulas is surgical excision, with conservation of as much lung tissue as possible in order to preserve pulmonary function, especially if multiple lesions are present. Surgery is usually curative. It is recommended even in the asymptomatic case since the risk of brain abscess, fatal hemoptysis or development of cerebral thrombosis when polycythemia occurs is far greater than the morbidity and mortality of lobectomy or segmental resection.

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DISSECTING ANEURYSM OF THE THORACIC AORTA: ITS ROENTGEN RECOGNITION*

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FROM its earliest description in 1761 by Nicholls,[11] dissecting aneurysm has captured the interest of the medical profession. Originally it was merely a pathologic curiosity. In 1863 the condition was first firmly established as a clinical entity by Peacock.[12] It has only been during the past few decades that increasing accuracy in making the diagnosis in the living patient has been evident. At the present time the clinical findings upon which the diagnosis can be made have been well documented.[5,13,16,20] In many instances the history, physical examination and laboratory findings are quite sufficient to establish it, but occasionally differential diagnosis from coronary occlusion, pulmonary embolism, a variety of neurologic disorders, pneumothorax or various forms of acute abdominal emergencies may be difficult or impossible. Electrocardiograms at times may be suggestive of myocardial infarction.[1,5] The condition most often ends fatally in a few hours or days, but in about 10 per cent the initial attack may be survived, and the individual may live for several months or even years. Jones and Langley[9] reported 2 patients who lived three and eight years respectively after evidence of the initial attack. This latter group, sometimes referred to as "healed" cases, at autopsy usually demonstrates a re-entry of the dissected hematoma back into the true lumen of the aorta or the iliac arteries. According to Jones and Langley, 96 per cent of patients who do not show this type of re-entry of the dissection into the aorta die in the first five weeks, while of the cases in which re-entry has been established, only 28 per cent died in that time. This fact has lead certain surgeons[7,15] to attempt to create an artificial point of re-entry into the aorta thus, in effect, producing "healing" of the dissection. More recently DeBakey and his associates[2] have successfully removed or repaired dissecting thoracic aneurysms in 9 individuals. Dissecting aneurysm has joined the list of conditions hitherto considered as interesting pathologic entities but which now has become of practical importance and must be recognized promptly.

PATHOLOGY

Sailer[14] has given a very complete discussion of the history and pathogenesis of this disease which will be summarized only briefly here. The condition more commonly occurs in males in a ratio of about two to one and usually in those over the age of forty. It has been described, however, in the very young (in a fourteen month old infant and in a ten year old boy[4]); the oldest recorded case was that of a woman nearly one hundred years old.[17]

It is now the commonly held view that the process begins as a degenerative one in the media of the aorta, usually at the junction of the middle and outer thirds. Cyst-like areas form and become filled by amorphous debris. In the diseased portions hemorrhage may take place—these hemorrhages probably occurring from the vasa vasorum. The coats of the aorta become separated by coalescence of these areas of degeneration and hemorrhage, the process usually extending throughout a considerable portion of the aorta. Associated with this process of medial degeneration and hemorrhage, transverse tears may occur in the intima; they are not, however, an invariable accompaniment of the disease, but most often occur in the supravalvular portion of the aorta and in the region near or

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just beyond the origin of the great vessels at the arch near the ligamentum Botalli. The dissection may begin in the arch or close to the aortic valve and will then, as a rule, progress distally; occasionally the process is retrograde. It is believed that when a tear takes place in the intima, blood under systemic pressure is permitted to enter the area of dissection, thus further increasing and accelerating the process of dissection between the walls of the aorta. Many cases promptly terminate by rupture of the dissection externally, most commonly (about 70 per cent[10]) into the pericardium. Other sites of hemorrhage are into the pleural spaces, mediastinum, the peritoneal cavity, retroperitoneal tissues, and other less common locations.

In about 10 per cent of cases a second tear in the intima occurs distally, not infrequently in the region of the bifurcation of the aorta. In these "healed" cases, under favorable circumstances, dissections in the aortic wall may become endothelialized and even calcified, and the individual may live several years.

Arteriosclerosis[10] may be seen in association with some dissecting aneurysm, but it *per se* does not play an important part in the classic case. The intimal tears, usually transverse, do not occur characteristically near areas of arteriosclerosis. Dissection of the aorta may be present with arteriosclerosis, but this is uncommon in the thoracic aorta and is usually much more limited in extent than in the classic case of dissecting aneurysm.

Hypertension is frequently, although not invariably, associated with dissecting aneurysm. Syphilis is not considered to be a causative factor.[14]

CLINICAL FINDINGS

The clinical features of dissecting aneurysm have been well discussed by a number of writers,[3,5,14,19] the majority emphasizing its sudden onset with a shocking, tearing pain in the chest either anteriorly or posteriorly. This pain characteristically migrates to the back and down into the abdomen and even to the legs. There is often a direct relationship between the location of the pain and the site of the dissection.[10] The severe pain is accompanied by great prostration and is not relieved by the usual amounts of medication. Fever and leukocytosis occur as the process of dissection advances. Various important arteries to the extremities, head and neck, chest and abdominal wall, kidneys and bowel may be involved or occluded. A great variety of clinical patterns may emerge.

Clinical differential diagnosis must include myocardial infarction, cerebral embolism,[18] pulmonary infarct, spontaneous pneumothorax, mediastinal emphysema, and a wide range of acute abdominal emergencies.

ROENTGEN FINDINGS

Although it has been generally agreed by most writers[13,19,20] that the roentgen findings may be of considerable help in the diagnosis of this condition, it has also been stated that they are not pathognomonic and that the diagnosis cannot be made or even suggested from the roentgen findings alone. Jackson and Slavin[8] have reported that only by angiocardiography can the roentgen diagnosis be established with certainty, and DeBakey and associates[2] consider this the most valuable diagnostic aid. Golden and Weens[6] in 1949 described the angiocardiographic findings in a single case of dissection of the thoracic aorta. In this, a thickening of the wall of the aorta beginning at the arch and measuring up to 26 mm. was demonstrated. Golden and Weens[6] thought that without angiocardiography it would have been impossible to differentiate this dissecting aneurysm from a simple dilatation of the aorta or an aneurysm of the cylindrical or saccular type.

The present series consists of 16 pathologically proved cases in which roentgenograms of the chest were available for review (Table 1). In 2 of these the aorta showed no change typical of dissecting aneurysm, al-

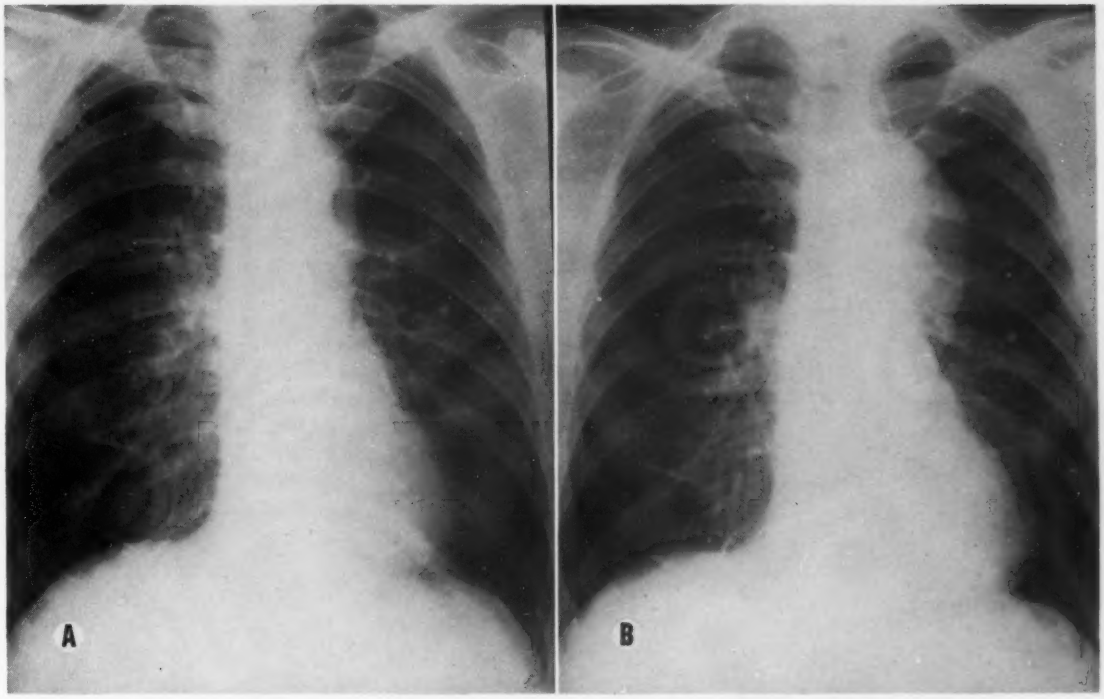

Fig. 1. Case 1. (*A*) January, 1953. Moderately tortuous aorta. (*B*) November, 1953. Aorta wider and slightly nodular. Heart somewhat larger.

though in each it was slightly elongated and tortuous. In a third case there was an aneurysmal dilatation of the aortic arch which could not be distinguished from a luetic aneurysm save by angiocardiography, which showed thickening of the aortic wall up to 2–3 cm. In 13 of the group the changes in the thoracic aorta were considered to be typical of dissecting aneurysm.

The most characteristic roentgenologic finding, and one on which most reliance was placed, was a definite change in the diameter of the aorta taking place between two successive examinations (Fig. 1, 2 and 3). This change was best recognized in the descending thoracic aorta where the vessel was outlined by air-containing lung on either side and was seen best usually in the lateral or left anterior oblique projections. At times, on heavily exposed films, the widened aortic shadow could be seen through the heart in the sagittal plane (Fig. 4).

A less common finding was increased prominence of the right border of the ascending aorta as it extended above and beyond the right heart border. In some instances this curve appeared angular and somewhat nodular as it emerged from the heart shadow on plain roentgenograms. Proof of widening of the ascending aorta could not be definitely established, although it could be inferred from this, since the opposite wall of the aorta at this level could not be distinguished from the remainder of the heart and great vessels. In a number of cases where the ascending portion was involved, the aorta approached more closely than is usual the posterior border of the sternum. Aortic tortuosity alone could also produce this change.

On satisfactorily exposed films, characteristically the widened, involved aorta displayed an obvious though not extreme irregularity in its outer diameter, producing an uneven, knobby appearance (Fig. 5). If serial roentgenograms were available, this abnormality was often quite apparent

TABLE I

DISSECTING ANEURYSM OF THE THORACIC AORTA

Case	Sex, Age	Pertinent History	Physical Examination	Blood Pressure
1	M 58	2–3 yr. hypertension. 3 mo. pain chest, sudden, severe rt. scapula to upper lumbar; substernal constriction. Continuous till hospitalization Nov., 1953	Rt. strabismus. Dilated neck veins. Cardiomegaly and hepatomegaly. Gallop rhythm. Tachycardia	270/170
2	F 75	3–5 yr. occ. nocturnal angina. Severe chest pain with collapse a few hours before admission 4.8.53	Shock. Distended neck veins. Pulmonary emphysema. Heart sounds distant. Liver and spleen large	70/—
3	M 68	Sudden acute mid-chest pain with dyspnea 24 hr., numbness and paralysis of legs. Adm. 6.8.52	Coarse rhonchi lungs. Abdomen distended, tympanitic, normal peristalsis	158/98 with marked variations
4	M 64	None beside suggestive roentgen findings of chest 2 yr. before admission—May, 1955. Hempotyses 3 mo. Second admission July, 1955 with interscapular pain	Essentially negative	270/130 60/40
5	F 61	Hypertension. 2-pillow orthopnea. Adm. 7.31.53 for surgery for diverticulitis	Essentially negative	200/110
6	M 72	Hypertension since 1937. 1 yr. low back pain. 8 mo. increasing dyspnea. 2 wk. marked increase dyspnea. Adm. 3.9.51	Dyspneic, cough, cyanosis; rales. Heart enlarged left	240/140
7	M 47	Rheumatic fever with stenosis. 24 hr. sudden excruciating pain mid upper abdomen, radiating back and arm. Adm. 4.8.51	Shock. Cyanotic. No palpable pulsations. Heart sounds very distant, of poor quality	Not obtainable
8	M 66	None. 6 hr. sudden severe pain across back, below scapulae, radiating to epigastrium. Adm. 10.9.48	Cheyne-Stokes respiration. Face florid. Legs cold, paralyzed	—
9	M 48	Hypertension. No history of syphilis. Epistaxes. 3 hr. lost blood by mouth. Adm. 11.28.52	Cold, clammy Cardiomegaly Op. Wiring aortic aneurysm	176/108
10	M 53	6 mo. repeated attacks of epigastric pain. 5½ wk. sharp pain left chest. Paralysis legs. Adm. 12.23.46	Venous distention. Loud rushing bruit legs and paravertebral area. Left chest dull to flat to percussion	170/100
11	F 75	Hypertension since 1951. Loss consciousness several hours. Adm. 9.8.55	Cyanosis. Cheyne-Stokes respiration. Partial paralysis. Heart large	210/120 140–100/0
12	F 69	5 hr. sudden severe epigastric pain radiating to back. Adm. 12.4.55	Not pertinent	140/100
13	M 36	7 yr. dry cough with nonradiating subscapular pain. 4 yr. pain moved to rt. Symptoms all worse. 2 yr. 25 aspirin tablets daily. Adm. 6.22.53	Tests for syphilis negative. Op. Resection thoracic aorta	Rt. 90/80 Lt. 120/90
14	F 22	3 yr. nephrosclerosis since 1951. 2 hr. chest pain Nov., 1953	Record not available	—
15	M 43	14 mo. hypertension. 1 wk. severe substernal nonradiating pain. Adm. 1951	Liver large. Left leg cold, weak pulse	220/140
16	F 74	5 yr. hypertension. 11 mo. increasing dyspnea. 4 hr. anterior chest pain	Neck veins pulsated 2 cm. above clavicle at 30° elevation. Apex beat in axilla. Gr. IV basal and precordial diastolic murmur and Gr. IV apical systolic murmur	200/80

Table I—(*Continued*)

Roentgenologic Findings	Course	Pathologic Findings
Jan., 1953—mod. tortuosity of aorta. No widening. Heart not enlarged. Nov., 1953—aorta wider from arch to diaphragm. Heart larger. Nov., 1954—no further change	Died Nov., 1954, in second acute attack	Healed communicating dissection aneurysm from arch to bifurcation
Ascending aorta prominent with haziness in ascending and descending portions. Heart enlarged	Died April 9, 1953	Aneurysm ascending aorta with dissection along pulmonary vessels, pleura and about mouths of coronary arteries
Ascending aorta slightly prominent; arch wide. Descending aorta fuzzy and wide as seen through heart	Died June 12, 1952	Dissecting aneurysm beginning just below arch and extending to level of renal vessels
1953—descending aorta tortuous and nodular suggesting aneurysm. May, 1955—descending aorta wider and again nodular. Ascending aorta mod. prominent. Heart larger. July, 1955—massive lt. pleural effusion	Died July 26, 1955	Dissecting aneurysm of descending thoracic aorta. Rupture into mediastinum and left pleural cavity
Widened irregular, nodular aorta. Heart large.	Died postop. Cerebral accident Aug. 23, 1953	Dissecting aneurysm, healed, extending throughout aorta to lower third abdominal portion
1944—min. aortic tortuosity; no widening. 1950—increased width from arch to descending port. with ascending more prominent, nodular. Lt. vent. large. 1951—no change 4 days before death	Died March 15, 1951	Dissecting aneurysm descending thoracic aorta, old, re-endothelialized. Rupture below subclavian artery and re-entry above celiac axis
Angular prominence ascending aorta. Fluid left pleural space. Left ventricular enlargement	Died April 12, 1951	Dissecting aneurysm ascending aorta extending to common iliacs with re-rupture into lumen and rupture into mediastinum
Arch probably wide and descending aorta fuzzy in outline. Not diagnostic except with good history	Died Oct. 10, 1948	Dissecting aneurysm with rupture into pericardium
Extensive widening of aorta from arch to diaphragm	Died 13 hr. postoperatively Nov. 29, 1952	Dissecting aneurysm, old, with perforation into bronchus right upper lobe
1946—great enlargement aortic arch. Increased prominence descending aorta, outline indistinct. Ascending aorta normal	Died Jan. 13, 1947	Dissecting aneurysm of descending portion of thoracic and abdominal aorta with rupture into left pleural cavity
May, 1955—heart large. Aorta tortuous, calcified, not wide. Sept., 1955—higher and wider. Desc. aorta wider; asc. more promin. and angular	Died Sept. 9, 1955	Dissecting aneurysm extending into great vessels of head and arms and down to iliac arteries
Both ascending and descending aorta prominent, indenting trachea at arch where it seems wide	Died Dec. 5, 1955	Dissecting aneurysm with rupture into pericardium and dissection along great vessels
Sharply localized dilatation in arch, indistinguishable from luetic aneurysm. Angiogram: Poor filling of a narrow channel in center of dilated portion of aorta. Wall 2–3 cm. diameter	Died during operation June 30, 1953	Dissecting aneurysm of aortic arch, old, double barreled
Aorta sl. tortuous but showed no diagnostic features and no change in 7 mo. period of time	Died Nov. 1953	Dissecting aneurysm of ascending aorta with rupture into pericardium
Aorta sl. tortuous and lt. ventricle sl. enlarged. No specific changes to suggest dissection	Died 1951	Dissecting aneurysm from subclavian to common iliacs, part. obstruction renals and part. occlusion lower abdominal aorta and lt. common iliacs
March, 1955—cardiomegaly and aortic dilatation. December, 1955—abnormally wide superior mediastinal shadow	Died Dec. 27, 1955	Dissecting aneurysm of descending portion of arch of aorta

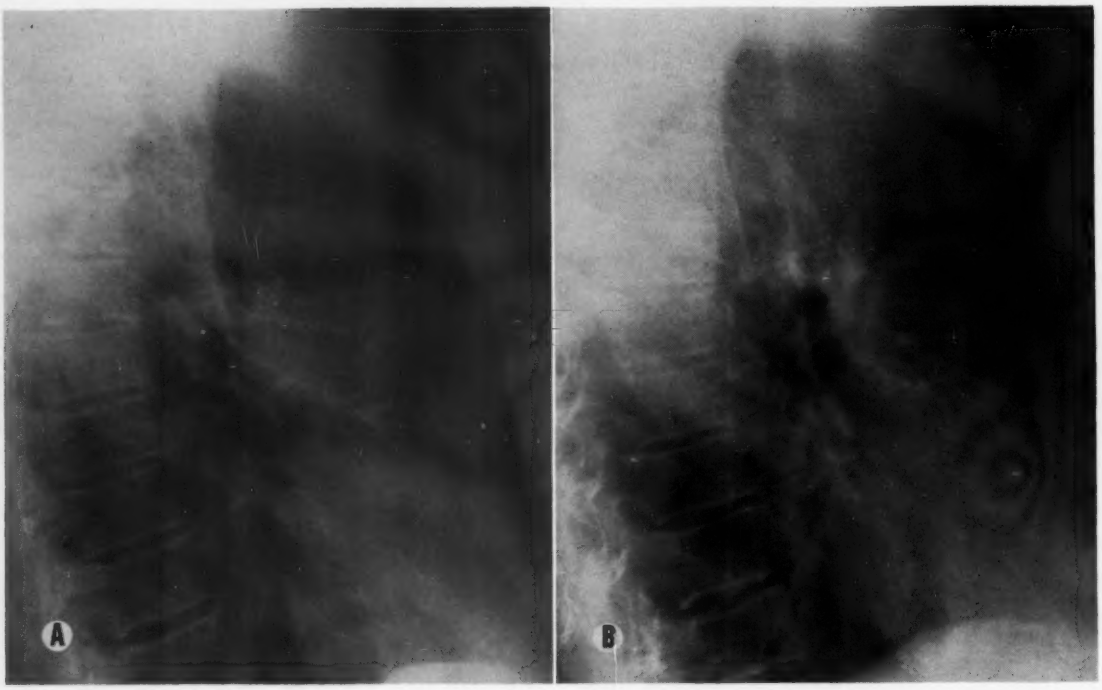

Fig. 2. Case 1. (*A*) January, 1953. Lateral roentgenogram demonstrates width of proximal descending aorta. (*B*) November, 1953. Increased width of the descending aorta.

and the diagnosis could be made with great assurance even without a typical clinical history. At times on a single examination changes were sufficient to suggest the diagnosis.

Since the majority of patients with dissecting aneurysm were acutely ill and pursued a rapid course to death, most examinations were technically incomplete and often unsatisfactory. In only a few cases exhibiting "healed" dissection could more complete and elaborate studies be

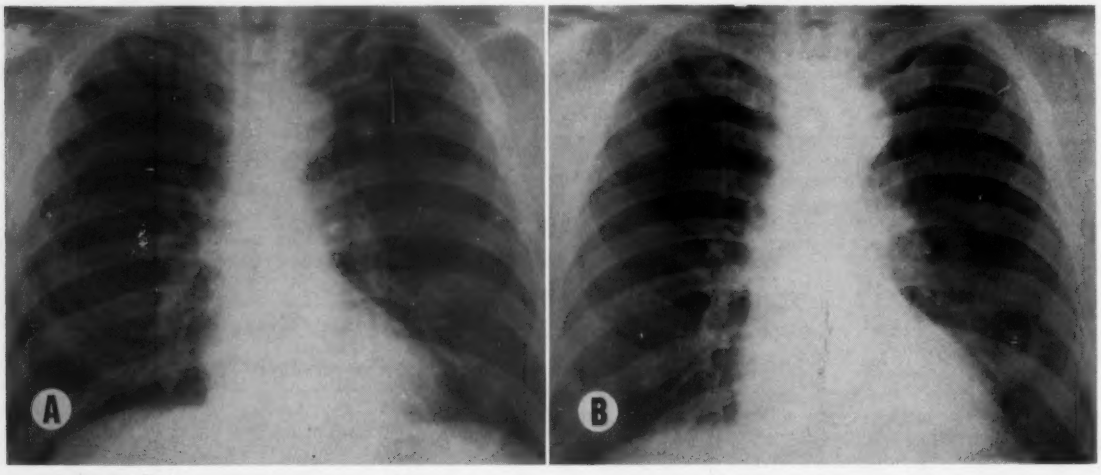

Fig. 3. Case 4. (*A*) 1953. Descending aorta is tortuous and slightly nodular. Left ventricle is somewhat enlarged. (*B*) May, 1955. Descending aorta is more prominent and nodular and is slightly hazy in outline. Left ventricle larger.

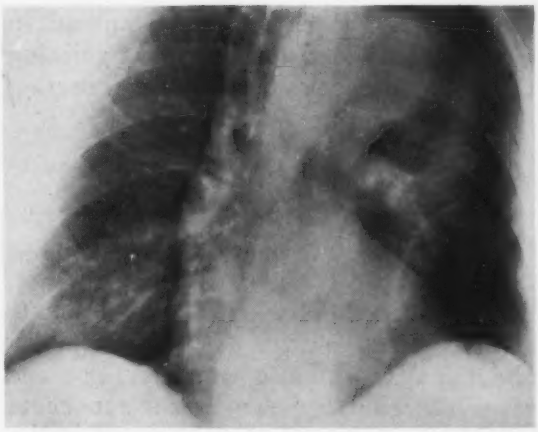

Fig. 4. Case 9. Heavily penetrated roentgenogram in sagittal view shows the widened descending thoracic aorta overlying the spine.

done. This series is too small to try to assess the value of fluoroscopy in the study of pulsation of the involved aorta.

It is well known that dissecting aneurysm may rupture externally into any of the adjacent structures in the thorax, abdomen, or retroperitoneal tissues. This rupture may be overwhelming, but more commonly a leakage from the aorta antedates the terminal episode. The latter was manifest in 7 cases, in all of which there were varying degrees of loss of the usual sharpness of outline of the aorta (Fig. 6). This produced a fuzzy appearance in the affected vessel, usually the descending thoracic, but at times the ascending portion. Occasionally the outline of the descending thoracic aorta was virtually lost, a finding which had been emphasized many years ago. Three patients with acute dissecting aneurysms showed fluid, presumably blood, in the left pleural space (Fig. 7); in one each, the hemorrhage was into the superior mediastinal structures, into the right middle lobe, and into the pericardium.

DIFFERENTIAL DIAGNOSIS

Arteriosclerosis, often associated with hypertension, produces elongation and increased tortuosity of the aorta but seldom results in significant dilatation. The outline of the vessel is usually smooth and clearly defined.

Arteriosclerotic aneurysms are commonly encountered in the abdominal aorta, rarely being seen in the thoracic portion. They produce as a rule a more localized, fusiform

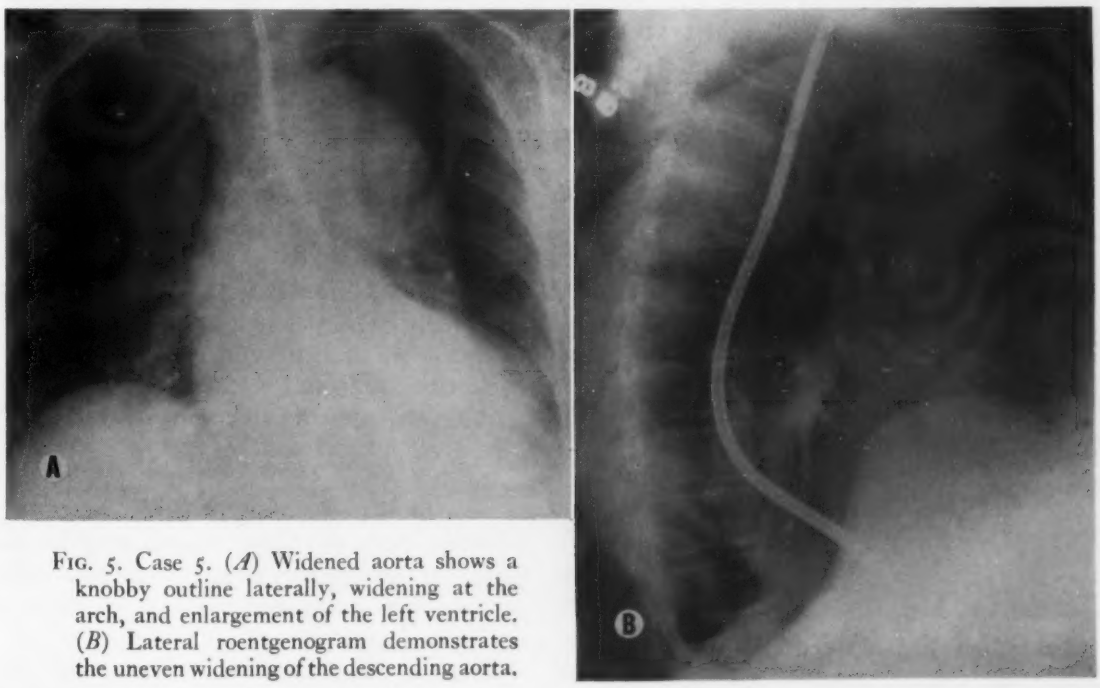

Fig. 5. Case 5. (*A*) Widened aorta shows a knobby outline laterally, widening at the arch, and enlargement of the left ventricle. (*B*) Lateral roentgenogram demonstrates the uneven widening of the descending aorta.

Fig. 6. Case 2. Both ascending and descending aorta are enlarged and show haziness of outline. There is left ventricular enlargement.

dilatation than the diffuse extensive widening seen with dissecting aneurysm.

Syphilis involves the ascending and transverse portions of the aorta causing a localized but often extreme degree of dilatation. If examined by angiocardiography, a luetic aneurysm usually shows a generalized widening of the lumen of the vessel with plaques of mural thrombi causing irregular narrowing. In contrast to this appearance, dissecting aneurysm involves the wall of the aorta more diffusely, albeit at times irregularly, producing a widening of the over-all diameter of the vessel, but at the same time narrowing its lumen. Angiocardiography[2,6,8] plays an important role in establishing the diagnosis, distinguishing the unusual localized form of dissecting aneurysm from the syphilitic.

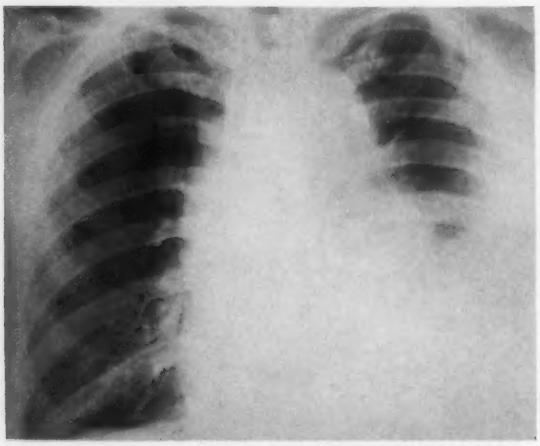

Fig. 7. Case 4. July, 1955. Massive left pleural effusion.

COMMENT

Most cases of dissecting aneurysm present with a rather typical history, but at times the clinical diagnosis may be obscure, being confused with a variety of acute cardiac, intrathoracic, neurologic or even abdominal conditions. Moreover, in some cases there may be no clear-cut acute episode to suggest the diagnosis clinically, and rarely there may be no history of any acute discomfort. It is true that the classic clinical picture is sufficiently diagnostic in itself to make roentgenographic examination unnecessary, but it is just as true, although not as well known, that the roentgenologic findings are usually quite typical and that the radiologist may be the first to suggest the correct diagnosis. In the acute case the importance of this latter possibility needs no emphasis since accurate diagnosis may forestall an unnecessary or harmful procedure. Now that surgical intervention is feasible, prompt and accurate roentgenologic recognition assumes great practical importance.

Angiocardiography will demonstrate most clearly the basic pathology, the thickening of the wall of the aorta, but this change may be inferred from the conventional roentgenograms with little likelihood of error if diffuse widening, particularly if this be irregular or "knobby" in appearance, of all or most of the thoracic aorta be present. Plaques of calcium in the wall of the aorta seen several millimeters from the outer margin of the vessel provide reliable evidence of thickening of the wall.[10]

SUMMARY

In a high percentage of cases dissecting aneurysm of the thoracic aorta may be recognized by conventional roentgenologic examination even without typical clinical findings.

Prompt identification of the condition

may decisively guide proper therapy, including operative intervention.

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RENAL ARTERY ANEURYSMS

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ANATOMY

IT IS reported that the majority of renal artery aneurysms occur in the main portion of the renal artery before it bifurcates or in an immediate major branch. Aneurysms involving the smaller arterial branches, that is intrarenal, are uncommon and the multilocular, intrarenal type is even rarer. Multiple or bilateral renal artery aneurysms are also very rare. An aneurysm of an accessory renal artery has been reported. It is of interest to note that of the proved cases reported in this paper one had a multilocular, intrarenal aneurysm (Case I, Fig. 1, 2 and 3); one had a saccular intrarenal aneurysm (Case II, Fig. 4); another had bilateral renal artery aneurysms, one of which was intrarenal and the other extrarenal (Case III, Fig. 5 and 6); and one had an extrarenal aneurysm involving a major arterial branch (Case IV, Fig. 7, 8, 9 and 10). Two suspected cases are of the extrarenal type (Case V, Fig. 11, 12 and 13 and Case VI, Fig. 14, 15, 16 and 17).

INCIDENCE

Renal artery aneurysm is a rare lesion as evidenced by the paucity of reported cases as well as the extremely low incidence in several large autopsy reviews with this lesion in mind. It occurs with approximately equal frequency in males and females and the majority of the patients are between the ages of forty and sixty years, although the youngest reported case was nine months of age and the oldest eighty-two years of age. Renal artery aneurysms are equally distributed in both kidneys. The recent evidence of decreasing incidence of false renal aneurysms is apparently due to an increasing awareness by physicians of this lesion and surgical intervention of true aneurysms before they have had an opportunity to rupture and form false aneurysms.

ETIOLOGY

The etiology of renal artery aneurysms is somewhat obscure. The following entities have been identified as causative agents:

1. (a) A congenital defect in the elastic tissue and media of arteries at their bifurcations as seen in intracranial aneurysms. This was apparently true in one of the cases reported in this series (Case II, Fig. 4).
   (b) Congenital arteriovenous aneurysm.
2. Arteriosclerosis, as in other locations, causing fusiform types of aneurysms (Case I, Fig. 1, 2, and 3 and Case IV, Fig. 7, 8, 9 and 10).
3. Syphilis causing the saccular type.
4. It has been suggested that severe systemic infectious or inflammatory processes may weaken the renal artery wall and predispose to the formation of aneurysms.
5. Periarteritis nodosa, although usually generalized, displays a particular affinity for the renal artery and has been identified as an etiologic agent in a few reported cases.
6. Tuberculosis has been identified as a causative agent in one reported case.
7. Rheumatic endarteritis has been identified as the cause in one reported case.

Trauma has not been, and probably never will be, entirely eliminated as a primary etiologic factor. The protective anatomy of the renal pelvis renders it extremely unlikely that trauma plays an important part in the primary etiology of true renal

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artery aneurysms. It assumes more importance as a secondary factor, particularly in the rupture of true aneurysms and subsequent false aneurysm formation. As in other diseases, a number of patients have a tendency to identify trauma as an etiologic agent and this would appear to be true of aneurysms of the renal artery.

PATHOLOGY

Renal artery aneurysms can be divided into two types, true and false. All of the proved cases (Cases I, II, III and IV) in this series were of the true type. True aneurysms are usually much smaller than the false type which can assume tremendous proportions. The true aneurysms may be saccular, which is the most common; fusiform, which is less common; dissecting, which is rare; and congenital arteriovenous which is very rare. The wall of a true aneurysm consists of the layers of the arterial wall and at least one of these remains intact. The false type of aneurysm represents a spontaneously controlled hemorrhage following the rupture of a true aneurysm or of the artery itself. The wall of a false aneurysm is composed of condensed fibrous tissue which forms around the extravasated blood and organized, laminated clot. This wall frequently becomes adherent to surrounding tissues. Both types of aneurysms often demonstrate typical calcification in their wall. False aneurysms may eventually rupture into the retroperitoneal space to form hematomata; hemorrhage extensively into the peritoneal space; erode into the collecting system of the kidney and lead to rapid exsanguination; dissect into and/or around the kidney substance producing varying effects; or rupture into renal veins. There may be obstruction of the renal artery with the development of a Goldblatt type of ischemic kidney leading to hypertension.

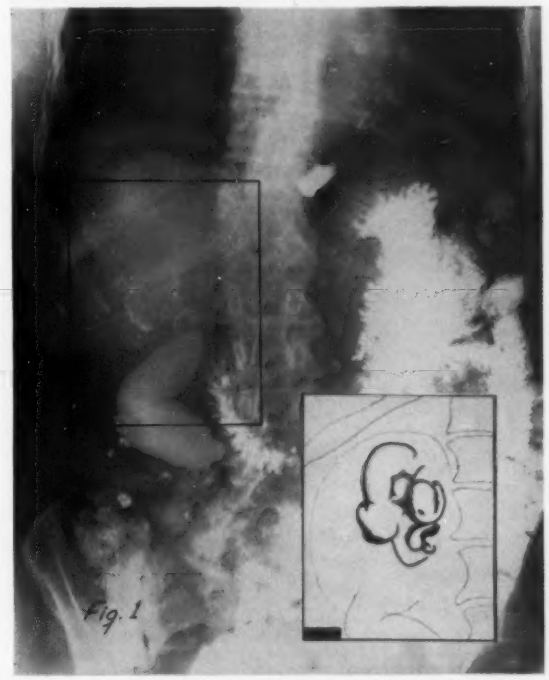

Fig. 1. Case 1. Irregular, multiloculated, vascular type calcification in the vicinity of the mid-portion and upper pole of the right kidney.

Fig. 2. Case 1. Retrograde pyelogram reveals marked displacement of the collecting system about this irregular calcified lesion.

CLINICAL EVOLUTION

True aneurysms are usually asymptomatic. The size of the aneurysm has some bear-

Fig. 3. Case I. Gross autopsy specimen containing a multiloculated, intrarenal aneurysm with a probe demonstrating its communication with a large branch of the right renal artery.

ing on this situation. False aneurysms may be asymptomatic, but this is contingent on the extent of the rupture, the rapidity of blood leakage, the distribution of this leakage as well as the location and size of the formed false aneurysm. In the symptomatic cases pain is common and is of the dull aching type located in the flank or upper abdomen. Hematuria often develops, particularly if there is direct communication with or erosion of the collecting system, and may be microscopic or gross leading to eventual exsanguination. In either true or false aneurysms, infarction of the kidney may result from occlusion of the renal artery or one of its branches and subsequently lead to hematuria. This may also occur with thrombi. Those cases with hypertension are usually associated with either occlusion of the renal artery or periarteritis nodosa. Very few hypertensive patients become normotensive following nephrectomy. A palpable mass may be present, obviously dependent on the extent of the lesion. It is usually fixed and does not move with respirations. Very rarely is there a palpable pulsation or an audible bruit, but when present they are pathognomonic. If signs or symptoms are increasing over a relatively short period of time, the prognosis is grave. The clinical picture of a false aneurysm often presents a difficult diagnostic problem. It may simulate rupture of the kidney, necrosis of a tumor, urinary calculi and other acute abdominal emergencies.

DIAGNOSIS

In most cases renal artery aneurysm has been noted and investigated in the course of examinations for other disease entities. Prior to the advent of arteriography, it was diagnosed at operation or necropsy. Plain roentgenograms and laminagrams can be suggestive, but it has been our ex-

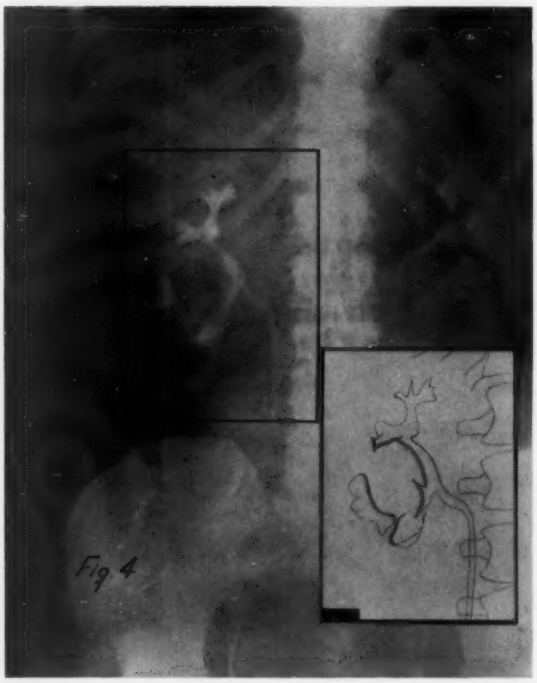

Fig. 4. Case II. Intravenous pyelogram reveals a typical mass lesion of the mid-portion and lower pole of the right kidney, markedly displacing the collecting system about it. There was no calcification demonstrated in this region.

perience that arteriography has been infallible in establishing this diagnosis as well as eliminating lesions simulating renal artery aneurysms. Plain roentgenograms usually demonstrate a fine wreath or ringlike, vascular type of calcification, the circumference of which is often incomplete where the aneurysm communicates with the lumen of the artery. This calcification may lie at any point in the course of the renal artery or its branches, but is more often medial to the upper third of the kidney and slightly anterior to the renal pelvis. Increasing size in this area of calcification is an important diagnostic sign. There may be an extrinsic type of deformity of the calyceal system and/or pelvis, and there may be displacement of the entire kidney. At times the diagnosis of a false renal artery aneurysm can be very puzzling, particularly if there is no calcification or the distribution of calcification is atypical. The lesions to be mentioned in the differential diagnosis are urinary calculi; calcified tumors, cysts

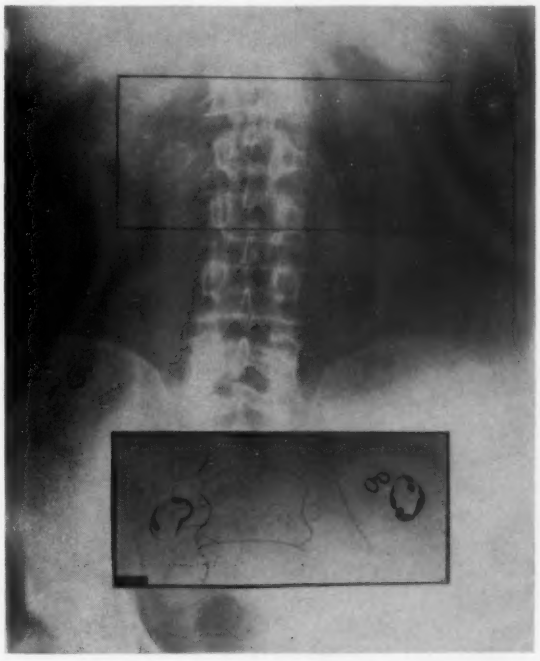

Fig. 5. Case III. Scout roentgenogram of abdomen reveals the typical vascular calcifications of renal artery aneurysms in the vicinity of the upper pole of the left kidney and the hilus of the right kidney.

Fig. 6. Case III. Aortography readily opacifies the renal artery aneurysms which are intrarenal on the left and in the hilus on the right.

(Case VII, Fig. 18, 19 and 20 and Case VIII, Fig. 24, 25 and 26) or abscesses, whether tuberculous (Case XIII, Fig. 33) or nonspecific (Case XII, Fig. 31 and 32); calcified lymph nodes; a calcified hematoma; calcification of autonephrectomy; gallstones (Case XI, Fig. 29 and 30); osteochondromata (Case X, Fig. 27 and 28); and other nonaneurysmal vascular calcifications (Case IX, Fig. 21, 22 and 23). Usually the calcifications in these lesions are not of the aneurysmal vascular type, that is, fine, circular and wreathlike, and they do not demonstrate the discontinuity in their periphery. In those cases where a differential diagnostic problem remains, arteriography readily establishes the diagnosis of aneurysm by opacifying the suspected calcification or mass and its communication with the vascular supply.

TREATMENT

Any true renal artery aneurysm which is symptomatic or shows signs of increasing in size and all false aneurysms whether symptomatic or not should be excised immediate-

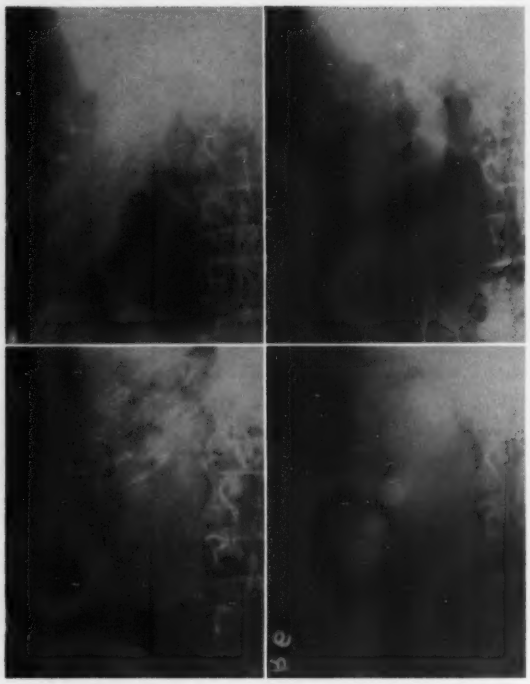

Fig. 7–10. Case iv. 7 and 8. Scout roentgenogram and a representative intravenous pyelogram reveal minimal dilatation of the renal pelvis with some interference with drainage in the erect position indicating some mild obstructive phenomenon at the ureteropelvic junction on the right. Several small rounded calcifications are noted in the vicinity of the lower pole of the right kidney and were thought to represent gallstones. 9 and 10. Six months later. Scout roentgenogram reveals a large irregular mass in the right upper quadrant displacing the proximal transverse colon inferiorly. The previously described calcifications are again noted and are in the vicinity of the mass although they are not characteristic of renal artery aneurysm. Intravenous and retrograde pyelograms on comparison with the previous study reveal rapid progression and an advanced degree of hydronephrosis, apparently due to an obstructive lesion at the ureteropelvic junction.

ly. The most important single reason for this rationale is the high frequency of sudden, fatal exsanguination. There are those who feel this is true in the asymptomatic cases as well, although most advocate careful observation and definitive surgery when and if they become symptomatic or show signs of increasing size.

If the aneurysm involves a small branch of the renal artery, one segment as in renal duplication or an accessory renal artery, it may be possible to excise it, repair the renal artery and still conserve all or a portion of the kidney. These alternatives are rarely possible and usually a nephrectomy is necessary.

PROGNOSIS

Some asymptomatic persons have lived for years before death occurred because of rupture or coincidental disease. In none of the cases reported in this series was the renal artery aneurysm the direct cause of death. The prognosis in unoperated lesions that are symptomatic or show signs of increasing size is extremely grave and death may be rapid. The usual cause of death is exsanguination from rupture of the aneurysm into the renal pelvis, retroperitoneal tissues or the peritoneal cavity.

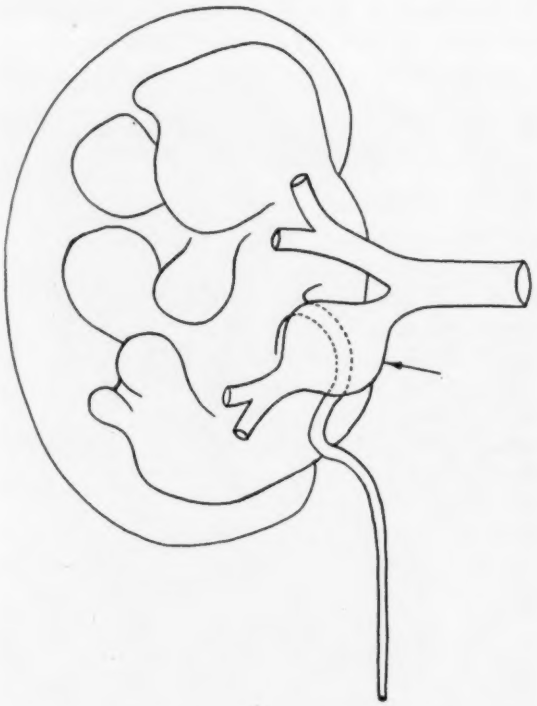

Fig. 10a. Case iv. Line drawing of operative findings. The renal pelvis and proximal ureter were literally "draped" over the large fusiform aneurysm of the right renal artery (arrow), causing obstruction and resulting in marked hydronephrosis.

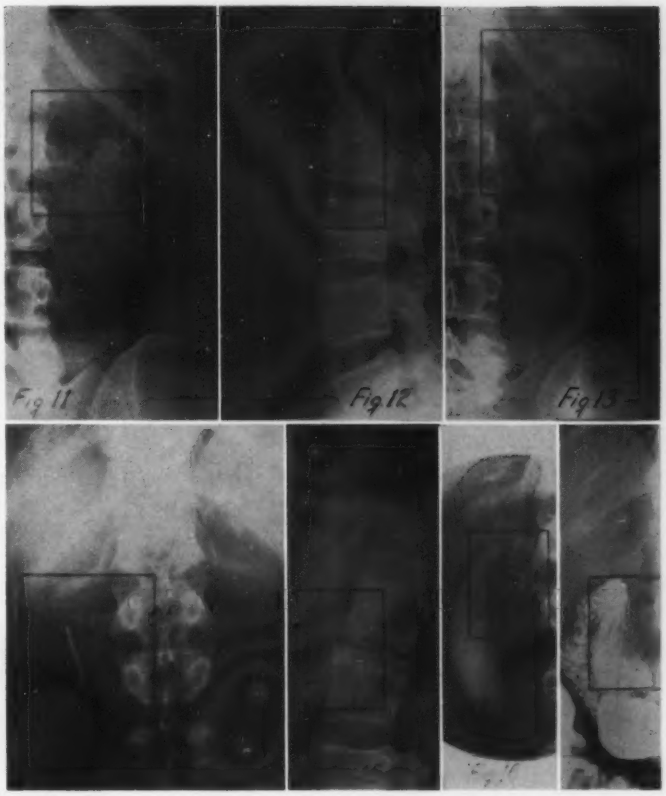

Fig. 11–17. Case v. 11, 12 and 13. Typical vascular type calcification of renal artery aneurysm medial to the upper pole of the left kidney with an incomplete circumference where the aneurysm communicates with lumen of artery. This is considered the most frequent location of renal artery aneurysm. Note the slight compression of the renal pelvis on the intravenous pyelogram. Patient refused aortography.

Case vi. 14, 15, 16 and 17. Typical location and vascular-type calcification of renal artery aneurysm in hilus of right kidney. This did not compress the renal pelvis on retrograde pyelography. It is of additional interest to note the larger vascular type calcification in the left upper quadrant which probably represents an aneurysm of the splenic artery. Cholecystography with excellent visualization of the cystic and common ducts and examination of the gastrointestinal tract readily revealed that this calcification did not involve these systems. Patient refused aortography.

REPORT OF CASES

1. *Proved Renal Artery Aneurysms.*

A. Case i. L.B. (Fig. 1, 2 and 3), a seventy-three year old woman, was admitted to Philadelphia General Hospital with a three-day history of passing bright to dark red blood per rectum at the time of defecation. There was a long-standing history of hypertension which was 190/104 on admission. There was a pulsating mass in the right upper quadrant with no audible bruit. The patient was unaware of this mass and apparently asymptomatic in reference to it. Roentgen studies (Fig. 1 and 2) revealed what was thought to be an intrarenal aneurysm of the right kidney and extensive diverticulosis of the distal colon. The patient had a positive serologic test for syphilis. Urinalysis was negative and the patient never developed hematuria. The patient continued to lose considerable blood by rectum and an exploratory laparotomy was done at which time a pulsating multicystic mass in the upper pole of the right kidney was palpated. The cause for the gastriointestinal bleeding was not determined. The patient expired within a few minutes following operation. Artificial respiration and cardiac massage were to no avail. Autopsy revealed the cause of death was pulmonary embolism and myocardial failure. The cause for the massive gastrointestinal hemorrhage was felt to be extensive diverticulosis and diverticulitis.

Gross pathologic examination of the right kidney (Fig. 3) revealed a multiloculated, well-calcified aneurysm occupying the upper pole of the right kidney with a readily demonstrable communication between it and a large branch of the right renal artery. There was advanced arteriosclerosis of the renal artery and its branches as well as the aneurysmal sac, and this was the etiologic factor in this case, despite the positive serology.

B. Case ii. E.G. (Fig. 4), a forty year old colored female was admitted to the Philadelphia General Hospital with a one-year history of persistent low back pain, burning on urination, frequency and nocturia of a mild degree. Blood pressure was 120/80, and no masses were palpable over the abdomen. The rest of the

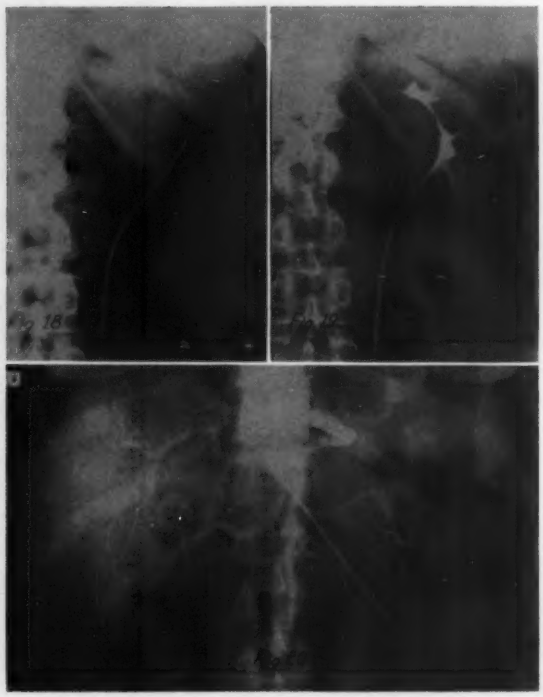

Fig. 18–20. Case vii. 18 and 19. Scout roentgenogram reveals an ill-defined mass lesion in the hilus medial to the upper pole of the left kidney with suggestive elliptical vascular calcification in its uppermost portion and marked displacement of the collecting system on intravenous pyelogram. In view of the location and appearance, it was deemed advisable to rule out renal artery aneurysm with possible rupture and false aneurysm formation. 20. Aortogram readily demonstrates this is not a vascular lesion. There is considerable superior displacement of the left renal artery due to this mass. In view of this, it was felt this most likely represented a partially calcified cyst in the hilus of the left kidney and this was confirmed at operation.

physical examination was entirely negative and the clinical diagnosis on admission was cystitis with pyelonephritis. An intravenous urogram demonstrated a well-circumscribed mass in the region of the pelvis and lower pole of the right kidney (Fig. 4). The roentgenologic diagnosis was cyst or tumor. No calcifications whatsoever were seen on her roentgenograms. Laboratory studies revealed a negative urinalysis and a positive serology (Kahn 8 units).

At operation the right kidney was removed. During the procedure palpation of the right renal artery revealed a definite bruit. Examination of the specimen disclosed a saccular aneurysm occupying the lower pole of the right kidney and communicating with a branch of the renal artery.

The wall of the saccular structure was composed chiefly of hyalin material and spindle-shaped cells, apparently smooth muscle and resembling arterial walls. A few small nodules or calcific deposits were present, but the appearance was not that of severe arteriosclerosis with aneurysm formation. There was no evidence of inflammation. A pathologic daignosis of aneurysm of the right renal artery, congenital, the latter being questionable, was made. The patient has remained asymptomatic and well.

C. Case iii. C. B. (Fig. 5 and 6), a forty-seven year old white man, was admitted to the Philadelphia General Hospital with long-standing hypertension, an old right-sided hemiparesis with flexion contractures of the extremities and a fairly recent left-sided hemiparesis following

Fig. 21–23. Case ix. 21 and 22. Scout roentgenogram reveals irregular vascular calcifications in the left upper quadrant probably lying in the splenic artery, but on intravenous pyelogram there is displacement of the calyceal systems and infundibula of the superior segment of the left kidney suggesting the possibility of an intrarenal renal artery aneurysm. 23. Aortogram readily demonstrates that all of the calcification is due to a tortuous splenic artery.

a cerebrovascular accident. Routine evaluation of this hypertension revealed calcifications in the vicinity of the renal pedicles which were felt to be bilateral renal artery aneurysms (Fig. 5). These were clearly demonstrated by means of aortography (Fig. 6). His laboratory studies, including urinalysis, were normal. Serology was negative. His course in the hospital was uneventful. In view of the bilaterality of these aneurysms, no definitive therapy has been recommended.

D. Case iv. C. S. (Fig. 7, 8, 9, 10, 10a), a seventy-eight year old white woman, was admitted to the Danville Memorial Hospital because of 3–4 day history of constant, dull right-flank pain, nausea and vomiting. Past history disclosed a similar episode six months prior to this adminission. Physical examination revealed a blood pressure of 180/90 and a large palpable, firm, nontender mass in the right flank. The rest of the history and physical findings were noncontributory. Urinalysis showed 3–4 red blood cells and 2–3 white blood cells per high power field but was otherwise negative. Blood serology was negative as were the other routine laboratory studies. Intravenous and retrograde pyelograms demonstrated marked right hydronephrosis, apparently due to an obstructive lesion at the ureteropelvic junction (Fig. 9 and 10). Review of the roentgenograms taken six months previously (Fig. 7 and 8) revealed similar findings of a minimal degree, indicating the chronicity and progression of this lesion.

At operation, a moderate-sized, intact, fusiform aneurysm was noted, involving the major arterial branch to the lower pole of the right kidney. This caused displacement and extrinsic pressure on the renal pelvis with inflammation and gradual fibrosis of the peripelvic tissues, resulting in marked obstructive hydronephrosis (Fig. 10a). The vessel was occluded by organized blood clot. The dissection and technical

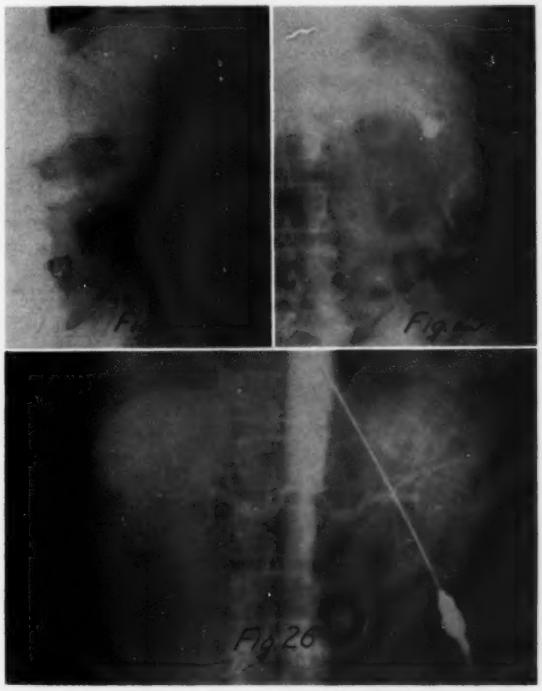

Fig. 24–26. Case viii. 24 and 25. Intravenous pyelograms show a mass with fine medial circumferential calcification, located in the hilus of the left kidney with compression and displacement of the renal pelvis. 26. Aortogram reveals some displacement of left renal artery superiorly but readily demonstrates that this is not a vascular lesion such as renal artery aneurysm. It was felt this represented a partially calcified cyst in the hilus of the left kidney.

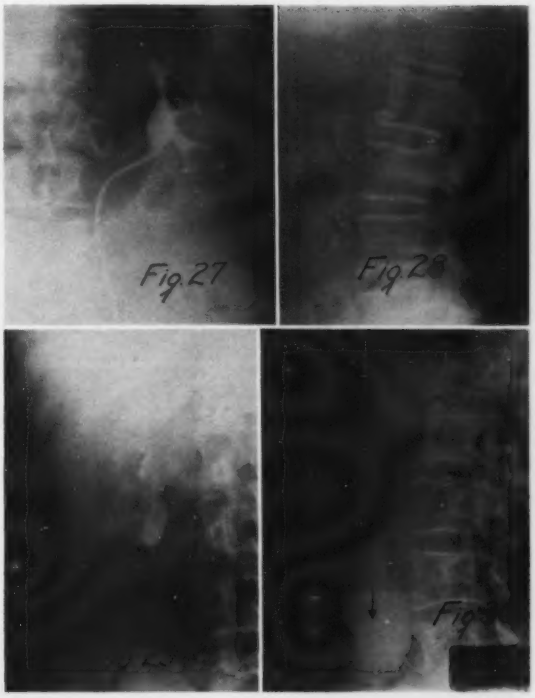

Fig. 27–30. Case x. 27 and 28. Autopsy revealed an osteochondroma which simulated renal artery aneurysm on intravenous pyelography.

Case xi. 29 and 30. Gallstone simulating a renal artery aneurysm on an intravenous pyelogram. Cholecystogram readily demonstrated this as an opaque calculus in the fundus of the gallbladder.

Fig. 31–33. Case xii. 31 and 32. Scout roentgenogram reveals a round, thick-walled area of calcification in the upper pole of the left kidney. Intravenous pyelogram shows some displacement of the superior calyceal system.

Case xiii. 33. Two rounded areas of calcification in right kidney representing tuberculous abscesses. Also, extensive involvement of left kidney was demonstrated on the intravenous pyelogram although the scout roentgenogram failed to demonstrate any calcification on the left.

difficulties on nephrectomy made it impossible to preserve the aneurysm. Pathologic examination revealed arteriosclerosis of the renal vessels and multiple thrombi within some of them.

2. *Suspected Renal Artery Aneurysms.*

A. Case v. C. B. (Fig. 11, 12 and 13), a forty-nine year old colored woman, was admitted to the Philadelphia General Hospital with complaints of backache and hypertension. Routine studies included intravenous pyelography which revealed a vascular type calcification in the vicinity of the left renal pedicle, probably representing an aneurysm of the left renal artery. Urinalysis revealed 3 plus albumin, 3 plus sugar and 3 to 5 white blood cells per high power field with many epithelial cells. There were no red blood cells in the urine. Patient refused aortography and surgery.

B. Case vi. M. M. (Fig. 14, 15, 16 and 17), a fifty-two year old white woman, was admitted to the Danville Memorial Hospital for repair of a rectocele and treatment of an acute exacerbation of chronic cervicitis. Roentgen studies revealed what appeared to be aneurysms of the right renal and the splenic arteries. The patient was asymptomatic in reference to the suspected aneurysms and refused aortography.

3. *Lesions Simulating Renal Artery Aneurysms.*

A. Case vii (Fig. 18, 19 and 20). Operation revealed a partially calcified benign cyst in the hilus of the left kidney.

B. Case viii (Fig. 24, 25 and 26). Patient refused surgery. The final impression was probable benign cyst in hilus of left kidney similar to Case vii, Figures 18, 19 and 20.

C. Case ix (Fig. 21, 22 and 23). Tortuous calcified splenic artery.

D. Case x (Fig. 27 and 28). Autopsy revealed an osteochondroma of lumbar spine.

E. Case xi (Fig. 29 and 30). Gallstone.

F. Case xii (Fig. 31 and 32). Operation revealed a calcified, nonspecific abscess of left kidney.

G. Case xiii (Fig. 33). Bilateral renal tuberculosis with positive smear, culture and guinea pig inoculation.

SUMMARY

Four proved cases, two suspected but unproved cases, and several cases of lesions simulating renal artery aneurysm are presented. Emphasis is placed on the use of aortography in the diagnosis of this rare lesion.

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ANEURYSM OF THE RENAL ARTERY*

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CRITERIA for the diagnosis of true renal artery aneurysm have been well documented in the literature; however, the diagnosis has been correctly established preoperatively in only 30 instances, as far as can be ascertained from the available literature.

It is the purpose of this paper to present a case of renal artery aneurysm diagnosed preoperatively on the basis of intravenous pyelography.

REPORT OF A CASE

A fifty-eight year old white female was seen by the Gynecological Service on March 1, 1955. A diagnosis of cystocele, rectocele and mild uterine prolapse was made; surgical repair was advised. Since the patient had concomitant symptoms referable to the lower urinary tract, *i.e.*, diurenal frequency, nocturia and moderate stress incontinence, a urologic consultation was requested. There were no other symptoms. There was no history of flank pain, chills or fever. The past history was not contributory.

On physical examination there were no abnormal findings except for the presence of a cystocele, rectocele and first degree prolapse of the uterus. The blood pressure was 140/80. The abdomen was soft. There were no masses. The kidneys were not palpable and there was no flank or costovertebral tenderness. There was no abnormal pulsation or bruit. The residual urine was 5 cc.

Cystoscopic examination on March 11 disclosed the presence of a moderate cystocele. The bladder was otherwise normal. The sphincteric action appeared normal; however, on sneezing and straining urine was noted to escape from the external urethral meatus.

Blood count, chemistries, blood serology and urinalysis were normal.

A plain roentgenogram of the abdomen showed normal renal outlines; an annular 2 cm. calcification was present in the region of the left kidney suggestive of aneurysm of the renal artery because of its appearance and location. Several phleboliths were noted in the region of the bony pelvis (Fig. 1*A*). An anteroposterior intravenous pyelogram showed the calcified ring-like mass to be intimately related to the hilus of the left kidney (Fig. 1*B*).

The fifteen minute film in the oblique position (Fig. 1*C*) showed a smooth compression defect of the mesial and anterior aspect of the left renal pelvis, apparently caused by the calcified mass. There was on the twenty minute film in a more oblique position also a slight indentation, made by the mass on the superior major infundibulum (Fig. 1*D*). In the lateral position the calcified mass maintained its close relationship to the left renal pelvis (Fig. 1*E*). The left kidney and ureter were otherwise normal. The right kidney, ureter and bladder were normal.

On the basis of these roentgenograms the diagnosis of aneurysm of the left renal artery was made. It was felt that other diagnostic procedures such as translumbar aortography, retrograde pyelography, and perineal air insufflation would be of only academic interest.

Operation was advised and was performed at Doctors Hospital on April 11, 1955. On exposing the left kidney through a lumbar flank incision, after resection of a segment of the twelfth rib, the renal pedicle was readily identified, isolated, and the aneurysm was easily exposed at the hilus; it was found to be located at the bifurcation of the main renal artery into its two main branches. The kidney was otherwise normal in appearance. Surgical excision or repair of the aneurysm was impossible because of its location. A left nephrectomy was performed. The surgical specimen was roentgenographed (Fig. 2), and photographed (Fig. 3).

Pathologic Report: Left kidney—the resected kidney measured 11×5×5 cm. The capsule was thin and stripped with ease leaving a tan brown slightly granular surface. At the hilus the renal artery showed a saccular aneurysm measuring 1.9 cm. in diameter. The aneurysm was located at the bifurcation of the renal artery into the upper and lower pole branches. On cut section the sac measured

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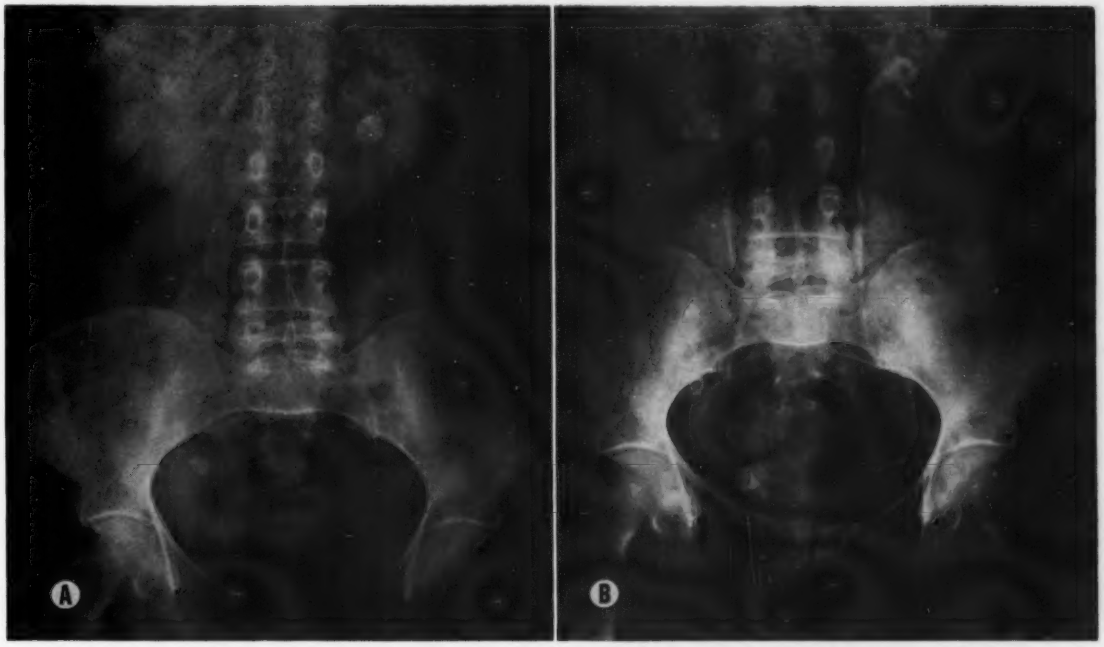

Fig. 1. (*A*) Plain roentgenogram shows annular calcification in the region of the left kidney. (*B*) Intravenous pyelogram—fifteen minute film, anteroposterior position. The calcified ring-like shadow is intimately related to the hilus of the left kidney.

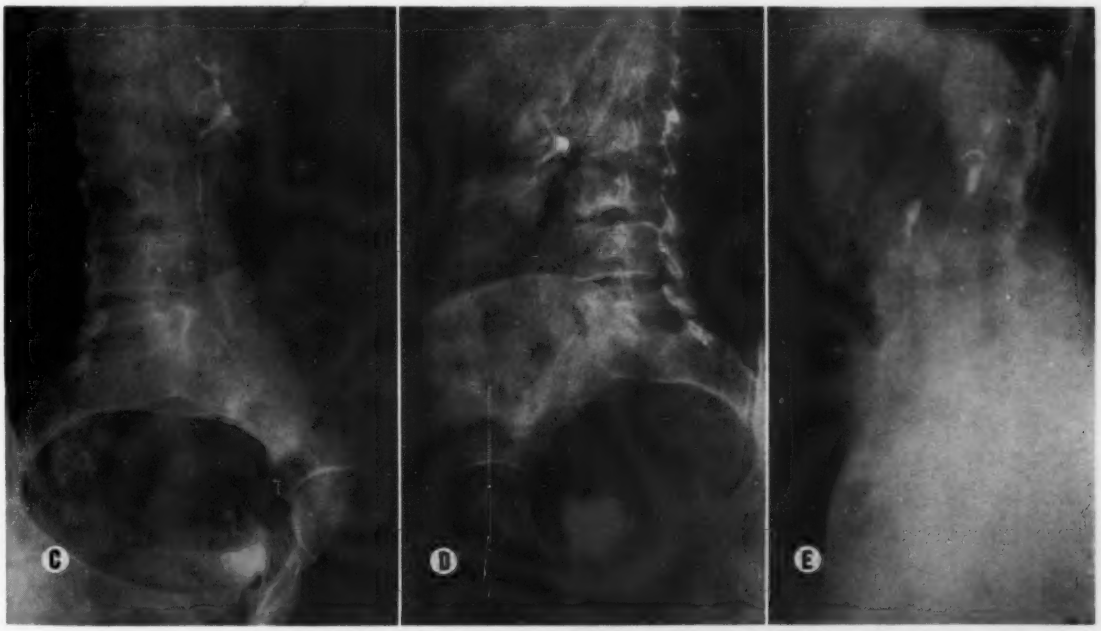

Fig. 1. (*C*) Intravenous pyelogram—fifteen minute film in the oblique position showing the calcified shadow maintaining its relationship to the renal hilus. (*D*) Twenty minute film in the more oblique position showing compression defect of the renal pelvis and indentation of the superior infundibulum caused by the calcified mass. (*E*) In the lateral position the intimate relationship between the calcified annular shadow and the renal pelvis is maintained.

FIG. 2. Roentgenogram of surgical specimen showing the calcified aneurysm.

1.7×0.8 cm. and was filled with clotted blood. The wall was calcific. The upper and lower branches of the renal artery opened into the sac, the larger opening being that of the lower branch. The pelvis was just inferior to the aneurysm and was distended. The ureter measured 3 cm. in length. The calyces were slightly dilated. The cortex and medulla revealed no features of note. The microscopic examination showed that the saccular dilation arose at the bifurcation of the renal artery (Fig. 4). The wall was thin. The intima was normal. The elastica was prominent and calcified. Involving the wall,

FIG. 3. Photograph of surgical specimen. Aneurysm arising at bifurcation of renal artery. Catheters are inserted into arterial branches.

FIG. 4. Photomicrograph of aneurysm showing its origin at bifurcation of main renal artery.

adjacent to the calcified internal elastica, there was a dense lamellar calcification. External to the calcified zone there was a thin, compact, hyaline layer. Inflammatory reaction was absent. The two branches of the renal artery were normal. The kidney, pelvis, and ureter appeared normal and were completely free of inflammatory reaction.

Diagnosis: Congenital aneurysm of renal artery with calcification.

The postoperative course was uneventful and the patient was discharged from the hospital on the ninth postoperative day.

SUMMARY

A case of renal artery aneurysm diagnosed correctly preoperatively has been presented. The diagnosis was made on an intravenous pyelogram.

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ADDENDUM

Several months following nephrectomy the patient underwent a vaginal hysterectomy with perineal repair. The recovery was uneventful.

The authors wish to thank Dr. Ernest B. Newman, Chief of the X-ray Department of the Hotel Health Center for his helpful interpretation of the roentgenograms.

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ANEURYSM OF HEPATIC ARTERY: ROENTGENOLOGIC FEATURES IN ONE CASE

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THE following case is being presented because of the extremely interesting features manifested from the roentgenologic differential diagnostic point of view, and because of the interest that rarity lends to any condition. At no time did the roentgenologist make the correct diagnosis, but it is felt that he does have a satisfactory reason for not having done so. Only findings pertinent to this study will be presented.

REPORT OF A CASE

In June, 1951, a sixty year old woman was registered at the Mayo Clinic with a chief complaint of "colon trouble." She had been quite well until six weeks before coming to the clinic, at which time diarrhea and abdominal cramps had developed with some loss of weight and appetite.

In her medical history it was noted that she had undergone appendectomy thirty-nine years previously and hysterectomy twenty-nine years previously. Physical examination revealed a pale woman with a fixed, palpable mass in the left upper quadrant of the abdomen. Roentgenologic examination of the colon showed a perforating carcinoma of the sigmoid. A few days later the carcinoma was resected. At the time of operation a small mass was found in the region of the gallbladder; this was thought to represent enlarged lymph nodes.

In June, 1952, she returned for a checkup, and evidence of recurrence was not detected.

Nine months later, on March 13, 1953, she experienced a sudden "gaseous" pain in the umbilical region while she was having lunch. This pain soon radiated up into her throat. She also felt weak and faint, and broke out into a cold sweat. She went to bed. That evening, and at times during the next day, she passed black stools accompanied by bright red blood. Three days later she returned to the clinic.

After a short preliminary period of rest and supportive therapy, the patient was sent for roentgenologic examination of the upper portion of the gastrointestinal tract. The only unusual finding was a shallow, smooth filling defect along the superior border of the first part of the duodenum (Fig. 1*a*). The roentgenologist assumed this deformity to be due to the gallbladder impression. It was therefore decided to make a further search for the source of the gastrointestinal hemorrhage, and a roentgenologic study of the small bowel was requested. At this examination, the defect in the duodenal cap had changed its aspect, and the roentgenologist now made a confident diagnosis of ulcerating leiomyoma of the duodenal cap (Fig. 2*a*). The remainder of the small bowel did not show evidence of any further abnormality.

On March 29, 1953, the patient was allowed to leave the hospital. The bleeding seemed to have stopped, but, on the same morning that she left, she had an internal hemorrhage and passed bright red blood in the stools. Exploration was carried out on the same day and an aneurysm of the hepatic artery was found closely adherent to the posterior surface of the duodenal cap. The hepatic artery was ligated proximal to the aneurysm. Four days later red blood drained from the duodenal tube. The patient was in shock. Eight days later the aneurysm was evacuated surgically and the hole in the duodenum was repaired. The patient died eleven days later of recurrent hemorrhage. Necropsy confirmed that the cause of death was fatal recurrent hemorrhage from an arteriosclerotic aneurysm of the hepatic artery.

* The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

COMMENT

Both roentgenologic diagnoses in this case were justified. Figure 1*b*, taken from a patient with gallstones, demonstrates the close relationship that the gallbladder may have to the duodenal cap. The second part of the duodenum of a patient having ulcerating leiomyoma is shown in Figure 2*b*. On the roentgenogram the small ulcer crater is demonstrated at the summit of the tumor, and the resemblance to Figure 2*a* is obvious. In retrospect, it is clear that the ulcer crater in Figure 2 is due to the aneurysm ulcerating into the duodenal cap, and that this was the site of the gastrointestinal hemorrhage.

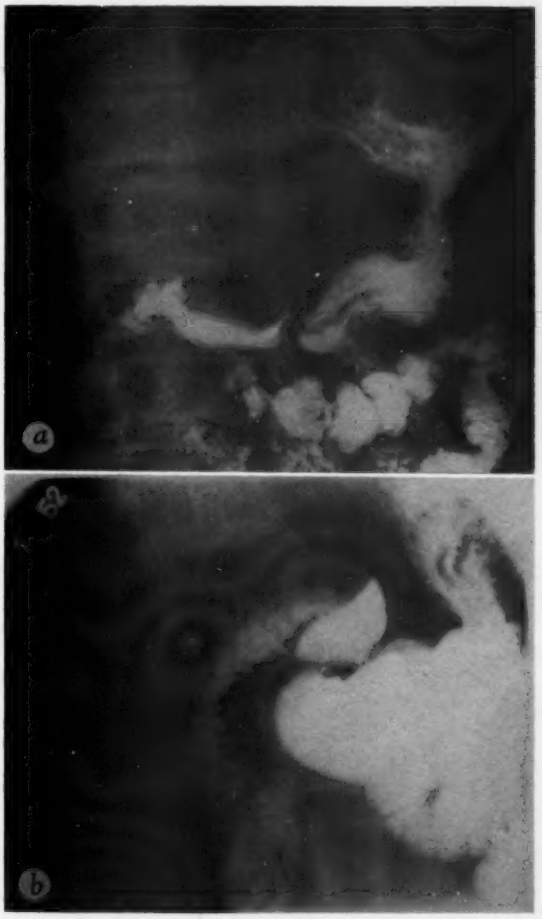

Fig. 1. (*a*) Smooth filling defect in the superior aspect of the duodenal cap which was thought to be impression of gallbladder. (*b*) Roentgenogram from a different patient, to demonstrate impression made on the duodenal cap by a gallbladder containing stones. Such a close relationship between the duodenal cap and the gallbladder is not infrequent.

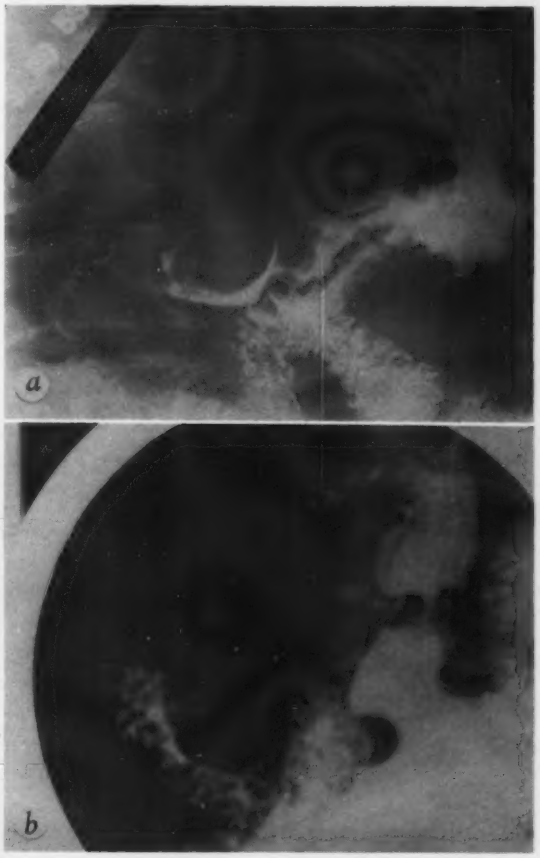

Fig. 2. (*a*) Roentgenogram of the duodenal cap taken three days after that in Figure 1*a*, showing defect in duodenal cap thought at this time to be due to an ulcerating leiomyoma. (*b*) Roentgenogram of the duodenal loop of another patient. The loop is deformed by an ulcerating leiomyoma which was subsequently resected. Note the similarity to the defect seen in Figure 2*a*.

Aneurysms of the hepatic artery are uncommon. In 1950, Grant, Fitts and Ravdin[2] reviewed the subject and found 87 cases reported in the literature; they reported 2 of their own. Volini and Popper[7] reviewed the subject in 1948 and presented a case. In the discussion of their paper, Lichtenstein[5] referred to 85 reported cases. In these cases, the commonest symptoms and signs were: paroxysmal pain in the right upper quadrant or epigastrium (58 cases), hemorrhage (40 cases), obstructive

jaundice (36 cases); in 67 cases rupture had occurred into various organs or places including the abdominal cavity, extrahepatic ducts, duodenum, gallbladder, portal vein and stomach. Gordon-Taylor[1] cited the clinical triad regarded by Malloy and Jason[6] as characteristic of aneurysm of the hepatic artery; namely, pain, hemorrhage and jaundice.

We know of only one other report concerning the roentgenologic features of aneurysm of the hepatic artery.[3] Jarvis and Hodes stated, "Obviously its recognition will depend upon the presence of calcific debris within the wall of the aneurysm, or perhaps even within the hemorrhagic mass." We are of the opinion that the case which we report herein presents additional criteria. Furthermore, it seems possible, in view of the fact that aneurysms of the hepatic artery are frequently associated with obstructive jaundice, that intravenous cholangiography may add information. A further method of diagnosis in which the roentgenologist may play a part is abdominal aortography. Such a case is to be reported in the near future.[4]

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ARTERIOGRAPHIC MANIFESTATIONS OF PERIPHERAL OCCLUSIVE VASCULAR DISEASE*

WITH THE REPORT OF TWO NEW SIGNS

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OCCLUSIVE peripheral vascular disease has been the main field of application for arteriography. A great wealth of literature on arteriographic experiences in this group of diseases is witness to this interest.[1,3,5,6,9,13,14] Most of the reports have been concerned with the study and detection of the site of obstruction and with the technique of the procedure itself.

The purpose of this communication is to present our experiences with peripheral occlusive vascular disease and to correlate the arteriographic findings with the underlying pathologic process. In view of the progress in the surgical management of some types of occlusive vascular disease, information obtained by arteriography about the status of the vascular bed beyond the obstruction assumes increasing importance. The status of the small vessels and the abundance and location of collaterals not only help in the selection of patients and the planning of the procedure but also to a certain extent allow some prognostication.

CLINICAL MATERIAL

The material presented is based on 84 femoral arteriograms. The youngest patient was thirty-one years old, the oldest ninety-six (Table 1). There were 65 males in the group and 19 females. By far the greatest number of patients were in the seventh decade of their life; however, there were 16 patients who were younger than fifty-five. Seven of these patients had uncomplicated advanced arteriosclerosis. Nine of these younger patients had diabetes or hypertension. Hypertensive patients were those who had a diastolic pressure at rest of at least 110 mm. Hg and a systolic pressure of at least 180. Two patients were younger than thirty-five. One had hypertension, the other long-standing diabetes. Of the 84 patients 27 had diabetes, 14 had hypertension, and 42 had uncomplicated arteriosclerosis. One patient had syphilitic vascular involvement.

All patients had intermittent claudication and a few had pain at rest. Thirty-five of the 84 patients (41 per cent) were eligible for vascular reconstitution operations.[7,10] In 33 a femoral-popliteal graft could be performed, and in 2 a bypass of the popliteal occlusion could be instituted.

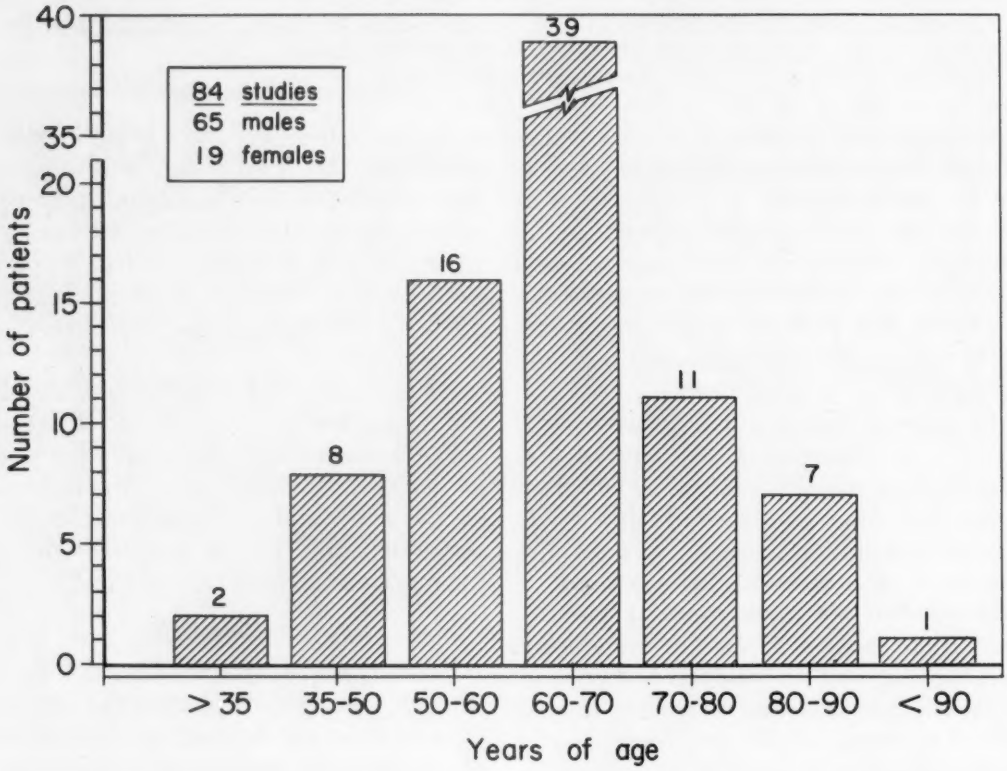
TECHNIQUE

The technique, described more fully elsewhere,[11] consisted of placing the entire extremity over the central, unshielded third of a tunnel box cassette holder. The entire cassette holder is 21 inches wide and 34 inches long. The two lateral thirds are shielded with lead. Two 14 by 17 inch cassettes are placed in the tray so that one half of the film is exposed while under the extremity and the other half is shielded. After the contrast material is injected the first exposure is made, followed in two seconds by the second exposure. Prior to the second exposure, the first half of the film is shielded as the cassette tray is pushed all the way into the cassette holder. The two-second interval between the two exposures is longer than we have been using in the demonstration of arteriovenous communications or bone tumors[8] and is necessitated by the slower flow in occlusive disease. The roentgen tube is elevated in such a fashion that the entire lower extremity from the

* From the Departments of Radiology and Surgery, University of Minnesota Medical School, Minneapolis, Minnesota. Presented at the Fifty-seventh Annual Meeting of the American Roentgen Ray Society, Los Angeles, California, September 25–28, 1956.

TABLE I

Femoral arteriograms in patients with symptomatic occlusive vascular disease



groin to the ankle is exposed at once. In order to compensate for the difference in thickness between thigh and leg, the heel effect is used on the thigh, and the cassettes are equipped with screens varying in intensification. Until recently we have been using 35 per cent iodopyracet* (diodrast), but lately we have used exclusively 35 per cent renografin.† This latter contrast medium is advantageous because arteriography may be performed under local anesthesia without discomfort.

ROENTGEN FINDINGS

General. All arteriograms were reviewed independently from the clinical history. Correlations with history and the clinical diagnosis were made only after the arteriographic findings were recorded. Every major vessel was analyzed separately and its appearance recorded, occlusions were localized and collaterals identified. Combined occlusions of several vessels were correlated with the underlying disease.

a. *Occlusions.* Table II lists the incidence of occlusions of various major vessels as

TABLE II

WHOLE GROUP. 84 STUDIES

Sites of Occlusion	No.	Per Cent
Superficial femoral artery	50	59
Profunda femoris	5	6
Popliteal artery	24	29
Tibioperoneal trunk	15	18
Posterior tibial artery	30	36
Peroneal artery	22	26
Anterior tibial artery	37	44

* Wintrop Stearn.
† Squibb.

encountered in our study. As was expected, the superficial femoral artery was found to be occluded most commonly. It was surprising to find as high an incidence of popliteal artery occlusions. The difference in the incidence of superficial femoral artery and popliteal artery occlusion when compared with the impressive report by Milanes *et al.*[9] may be due to the selection of patients. All of our patients had intermittent claudication, while these authors arrived at their material by selecting arteriograms from their collection only if occlusions were shown, if the study was technically good and if there had been a follow-up. Arteriography was performed on the majority of the patients that came to their vascular clinic. In our study, as theirs, the incidence of occlusions in the leg was high; however, the peroneal artery was much more frequently occluded in our series. In Lindbom's classic report[6] the peroneal artery was even less frequently occluded, and the posterior tibial artery was more frequently occluded than the anterior tibial artery. As seeen in Table II in our series, the anterior tibial artery was somewhat more frequently involved. The profunda femoris was occluded in a surprisingly large number of cases. Lindbom, however, has found only 4 out of 108 cases in which the profunda femoris had occlusions. In 1 such case the profunda femoris was the only vessel in the thigh to be occluded.

In a large number of our cases, a vessel, although not completely occluded, showed severe narrowing and irregularity (Fig. 1). When these instances showing severe narrowing, which we interpret as impending occlusion, are added to the ones showing complete occlusion, the superficial femoral artery is challenged in frequency of involvement by the popliteal artery. In our series this latter proved to be the most frequent severely narrowed vessel without definite occlusion (Table III). As in Lindbom's series the superficial femoral artery was most frequently occluded in the Hunter's canal. It was the only vessel occluded in 8 out of 84 cases (9.5 per cent) and was closely rivaled by the popliteal artery which was the only vessel occluded in 5 instances (6 per cent). The superficial femoral artery was occluded and the popliteal artery open in 33 studies, while the superficial femoral artery was simultaneously occluded with the popliteal

FIG. 1. Femoral arteriogram of a patient with arteriosclerosis obliterans. There are several areas of severe narrowing of the lumen of the superficial femoral and the popliteal arteries. Note the tortuosity and lack of tapering of the collaterals.

TABLE III

WHOLE GROUP. EIGHTY-FOUR STUDIES. SITES OF SEVERE OCCLUSIVE INVOLVEMENT. VESSELS EITHER OCCLUDED OR NEAR OCCLUSION (GRADE 3 OR 4)

Site	No.	Per Cent
Superficial femoral artery	62	74
Profunda femoris	15	18
Popliteal artery	45	53
Tibioperoneal trunk	20	24
Posterior tibial artery	39	46
Peroneal artery	35	42
Anterior tibial artery	44	52

TABLE IV

THE DISTRIBUTION OF COLLATERALS WITH OCCLUSIONS IN THE THIGH

A. Superficial femoral artery occluded	
Popliteal artery patent (suitable for by-pass graft)	
Thirty-three studies	
Collaterals	Per Cent
Obturator artery	9
Inferior gluteal arteries	15
External pudendal artery	3
Superior geniculate artery	6
Medial circumflex femoris and medial perforators	6
Lateral circumflex femoris group	100
B. Superficial femoral artery occluded	
Popliteal artery occluded	
Seventeen studies	
Collaterals	Per Cent
Obturator artery	23
Inferior gluteal arteries	23
Superior geniculate artery	29
Lateral circumflex femoris group	89

artery in 17 cases (Table IV). Table V, which will be more fully discussed later, shows the incidence of occlusion of the leg vessels in studies in which the superficial femoral artery was open.

b. *Calcification.* Very little can be added to Lindbom's conclusions regarding calcification. Medial calcification was seen both in the leg and thigh, but in almost all cases it was not seen in the occluded area. The intimal type of calcification was usually seen either in an area of occlusion or of severe involvement. The vessel that most frequently showed this type of calcification was the peroneal artery. In 10 cases out of the 22 with occlusion of this area, there was calcification in the region of the peroneal artery. Even without complete occlusion, however, this vessel showed calcification more often than others, being followed closely in incidence only by the superficial femoral artery.

c. *Microaneurysms.* In a rather large percentage of our arteriograms, we encountered small outpouchings on the side of the vessels, usually on branches of the larger arteries of the thigh and leg. These outpouchings were studied first independently of the clinical information on the patients, but when correlated the interesting results shown in Table VI were obtained. We have called these outpouchings microaneurysms because of their appearance. They closely resemble such formations seen in the fundus of the eye in diabetes. The danger of calling a winding tortuous arteriosclerotic collateral a microaneurysm can be avoided. We have called microaneurysms only those structures that could be seen in their entirety to be separate from the underlying vessel, and when on two films no vessel was seen to continue from the microaneurysm itself as a branch of the underlying vessel. We have tried in vain to obtain microscopic histologic sections of these structures. They seem to collapse in tissue specimen and are demonstrable only on the films (Fig. 2 and 3). Figure 2 is the arteriogram of an injected amputated leg of a mild diabetic with severe occlusive disease. The injection was performed with a thin barium suspension. An excellent demonstration of the microaneurysms has been obtained. As with clubbing, which will be described in

TABLE V

SUPERFICIAL FEMORAL ARTERY PATENT, POPLITEAL AND ALL MAJOR LEG ARTERIES OCCLUDED. THREE STUDIES (ALL 3 IN DIABETIC PATIENTS)

SUPERFICIAL FEMORAL ARTERY PATENT	
POPLITEAL ARTERY PATENT	
ALL MAJOR LEG ARTERIES OCCLUDED	
Total 9 patients	
With diabetes	5
With hypertension	2
Arteriosclerosis obliterans	2

TABLE VI

PRESENCE OF MICROANEURYSMS

	Total	None	Rare	Frequent
Diabetic	27	0	6	21
Hypertensive	14	2	7	5
Arteriosclerosis obliterans	42	35	7	0
Syphilitic	1	1	—	—

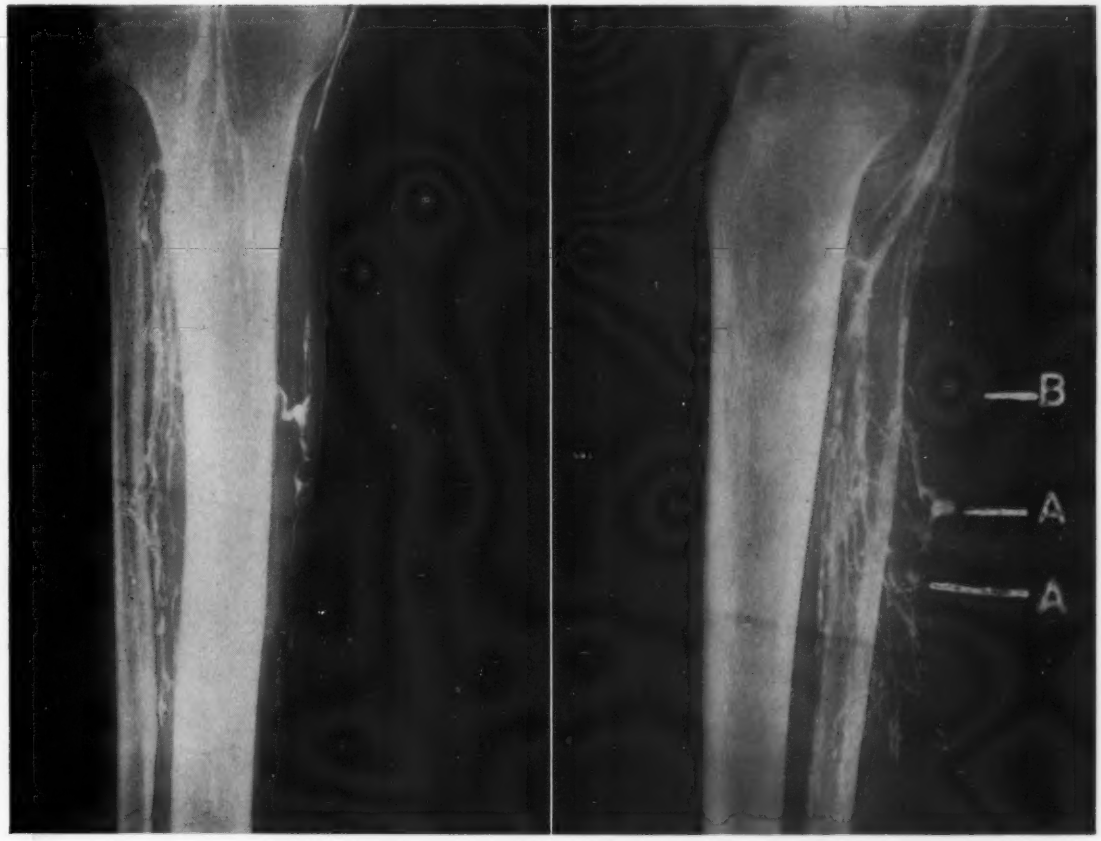


Fig. 2. Amputated limb injected with a thin barium suspension. Multiple small vessel occlusions are demonstrated. Many rather large microaneurysms (A) are present. There is considerable clubbing (B).

the next paragraph, the findings on the film are difficult to reproduce in the photograph.

d. *Clubbing.* In a large number of arteriograms, sudden endings of small vessels, sometimes quite close to their origin, were noted. Quite frequently, the ending of such a vessel had a small dilatation. It is for this reason that we applied to it the descriptive name of clubbing. Table vii shows the incidence of clubbing in our series and its correlation with the associated disease. We believe the clubbing is a manifestation of small vessel disease and that it can be correlated with the changes of endarterial intimal proliferation, which is encountered in this same type of vessel under the same conditions and which has the same appearance. The frequency of demonstration of clubbing by arteriography is not an adequate indication of its actual distribution in the limb as we believe that legs with poor collaterals and severe involvement of major vessels may have a significant number of occluded small vessels which we are unable to demonstrate. Figure 2, which shows the injected amputated limb, shows also numerous clubs. Figures 4 and 6 show clubbing in an arteriogram. One of the dangers in evaluating clubbing is to be satisfied with its demonstration on only one film, as it is

Table VII

PRESENCE OF CLUBBING

	Total	None	Rare	Frequent
Diabetic	27	1	11	15
Hypertensive	14	1	5	8
Arteriosclerosis obliterans	42	33	8	1
Syphilitic	1	—	—	1

FIG. 3. Femoral arteriogram of a diabetic patient showing microaneurysms (A). A cluster of clubbing is also seen (B).

FIG. 4. Femoral arteriogram of a diabetic patient showing a popliteal artery occlusion, microaneurysm (A) and clubbing (B).

possible to see the vessel filled beyond the fictitious occlusion on the next exposure. In the study of changes in small vessel disease, serial arteriography is preferable to our method, which is a compromise.

e. *Collaterals*. Table IV shows the principal collateral channels carrying the blood to the thigh and leg when the superficial femoral artery is occluded and the popliteal artery is patent or when both of these major vessels are occluded. It is important for the radiologist to inform the surgeon of the location of the majority of the large collaterals. The lateral circumflex branches of the profunda femoris are the most frequent and important collateral branches in both instances. Medial perforators and the medial circumflex femoral artery rarely constitute the main collateral vessels, but it is of importance to recognize when this condition exists (Fig. 5). Table VIII shows the incidence of poor or abundant collaterals correlated with the clinical diagnosis. It is noted that patients with uncomplicated arteriosclerosis have the highest incidence of a good collateral bed. Patients with diabetes have the poorest. Ross and Keele[13] have shown that if collaterals are adequate many patients who have vascular occlusions will remain undetected. In their series 7 out of 100 unselected asymptomatic patients had gross arterial occlusions. It is possible that the high incidence of patients with poor collateral circulation in uncom-

TABLE VIII

STATUS OF COLLATERALS

	Total	Poor	Good
Whole group	84	38	46
Arteriosclerosis obliterans	42	14	28
Diabetic	27	17	10
Hypertensive	14	6	8
Syphilitic	1	1	—

plicated arteriosclerosis in our group is due to the fact that all of our patients were symptomatic.

The collateral vessels seen with occlusion of arteries in the leg were not as predictable as with occlusion in the thigh. The majority originated from muscular branches of the peroneal artery, but atypical channels originating from the inferior geniculate arteries or the other leg arteries were quite common.

Arteriosclerosis. Forty-one of our patients formed a group in which no other disease associated with arteriosclerosis could be demonstrated. One patient had hypothyroidism and cholesterolemia but has still been added to this group. Table IX shows the incidence of the site of occlusion in this group. A comparison with Table II shows little difference, except for a higher incidence of popliteal artery occlusions. The occlusion of the large thigh vessels is much more frequent in this group than in the patients who also have diabetes. From Tables VI, VII and VIII it is to be stressed that this

FIG. 5. Femoral arteriogram of a ninety-six year old male with arteriosclerosis obliterans. There is an occlusion of the superficial femoral artery. Lateral circumflex femoral collateral (A). Medial circumflex femoral collateral (B). Clubbing (C).

TABLE IX

UNCOMPLICATED ARTERIOSCLEROSIS OBLITERANS. 42 PATIENTS

	No.	Per Cent
A. Sites of complete occlusion		
Superficial femoral artery	31	74
Profunda femoris	2	4
Popliteal artery	12	29
Tibioperoneal trunk	12	29
Posterior tibial artery	13	31
Peroneal artery	12	29
Anterior tibial artery	17	40
B. Sites of severe involvement or occlusion		
Superficial femoral artery	35	84
Profunda femoris	11	26
Popliteal artery	24	57
Tibioperoneal trunk	14	33
Posterior tibial artery	19	45
Peroneal artery	18	43
Anterior tibial artery	20	48

group has a high incidence of good collaterals and a low incidence of clubbing and microaneurysms. Frequent microaneurysms were not seen in any case of this group, and only 1 case had frequent clubbing (Tables VII and VIII).

TABLE X

PATIENTS WITH DIABETES (27)

	No.	Per Cent
A. Sites of complete occlusion		
Superficial femoral artery	10	37
Profunda femoris	2	7.5
Popliteal artery	4	15
Tibioperoneal trunk	2	7.5
Posterior tibial artery	11	41
Peroneal artery	6	22
Anterior tibial artery	13	48
B. Sites of severe involvement or occlusion		
Superficial femoral artery	15	55
Profunda femoris	3	11
Popliteal artery	12	44
Tibioperoneal trunk	3	11
Posterior tibial artery	14	52
Peroneal artery	11	41
Anterior tibial artery	14	52

It has to be admitted that this group is somewhat artificially and perhaps arbitrarily created as the patients with diabetes, hypertension and syphilis undoubtedly also had arteriosclerosis. However, the distribution of manifestations suggested this division.

We do not wish to enter into the controversy as to the entity of Buerger's thromboangiitis obliterans. In his classic work Buerger[2] introduced the term and stressed the inflammatory changes in the arterial wall. In their cases of chronic obliterative arterial lesions, Gery, Fontaine and Branzeu[4] found no changes that would fit Buerger's description, and they concluded that for all practical purposes they were indistinguishable from arteriosclerosis.

Lindbom believed that so-called thromboangiitis obliterans may be a disease of the blood, as also suggested by Nygaard and Brown.[12] In reviewing our cases in this group, there were none that had clinical or roentgenologic properties that would set them apart as Buerger's disease. Therefore, we have not been using this term in our study, although it could be applied to the 7 patients below the age of fifty-five who had advanced occlusive arteriosclerotic disease. The only classification of this group would be along the lines of the discussion by Greenwald *et al.*[5] who, on the basis of 103 femoral arteriograms, divided their cases into cases with intimal changes only, cases showing occlusion with refilling and without refilling at a lower level. In our group, the cases in which there was no filling of

TABLE XI

PATIENTS WITH DIABETES UNDER THE AGE OF FIFTY-FIVE (6)

Sites of occlusion	No.
Superficial femoral artery	1
Tibioperoneal trunk	1
Anterior tibial artery	4
Peroneal artery	4
Posterior tibial artery	6

Fig. 6 (*a*) Advanced occlusive disease in a patient with syphilis, showing poor collaterals. (*b*) Same patient. Note multiple areas of clubbing (A).

the distal parent vessel usually had poor collateral circulation.

The collaterals in this group showed no tapering and were very tortuous, running not only in centrifugal, but in all directions.

Diabetes. Twenty-seven studies were performed on patients with diabetes. The usual changes of arteriosclerosis obliterans were modified in this group. Table v shows that 55 per cent of the patients with both the superficial femoral and popliteal arteries open and all major leg vessels closed were diabetic. All 3 patients with all major leg vessels and the popliteal artery occluded and the superficial femoral artery open were diabetic. A disproportionate number of diabetics were also encountered in the younger age group. The distribution of the sites of occlusion in these patients is given in Table xi. The diabetic patients also predominated in the group with poor collaterals. Microaneurysms and clubbing were also one of the characteristics of this condition (Tables vi and vii), and microaneurysms were more frequently encountered in patients with diabetes than in those with hypertension. Table x shows the sites of complete occlusion and the sites of severe involvement or occlusion in the 27 patients. The occlusions in leg vessels actually outnumbered those in the superficial femoral artery.

Hypertension. There were 14 patients with hypertension. Table xii shows the sites of arterial occlusion. As in the group with arteriosclerosis obliterans, the thigh vessels are most frequently involved, but the number of patients with popliteal artery occlusion is high. Both clubbing and microaneurysms were seen in a large number of patients, but frequent clubbing was more common (Tables vi and vii). It was noted

TABLE XII

PATIENTS WITH HYPERTENSION (14)

	No.	Per Cent
A. Sites of complete occlusion		
Superficial femoral artery	8	57
Profunda femoris	0	0
Popliteal artery	7	50
Tibioperoneal trunk	1	7
Posterior tibial artery	5	36
Anterior tibial artery	6	43
B. Sites of severe involvement or occlusion		
Superficial femoral artery	12	85
Profunda femoris	1	7
Popliteal artery	9	64
Tibioperoneal trunk	3	21
Posterior tibial artery	6	43
Peroneal artery	6	43
Anterior tibial artery	10	71

that both the clubbing and microaneurysms in these cases were less frequent distal to the site of a major occlusion.

Syphilis. Only 2 patients with tertiary syphilis were studied.* Both patients also had syphilitic aortitis and aneurysms. Clubbing of multiple small vessels without microaneurysms was seen (Fig. 6). As the finding of clubbing would fit with the known pathologic histology of intimal proliferation in the vascular involvement of this disease, it is reported as an illustration of the nonspecific significance of clubbing.

SUMMARY

Eighty-four femoral arteriographic studies of patients with symptomatic occlusive vascular disease are presented. The sites of occlusion and the sites of collaterals are reported for the whole group, and the differences between patients with arteriosclerosis obliterans, and arteriosclerosis in conjunction with diabetes and with hypertension are pointed out. Microaneurysms and clubbing, which represent two manifestations of small vessel disease, are described and their incidence in the conditions studied is indicated.

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* One of these 2 patients was studied after completion of the tables and is not included in them.

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Vol. 78, No. 2

A CASE OF LINDAU'S DISEASE SIMULATING ANOREXIA NERVOSA*

A ROENTGENOLOGIC REPORT

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THE current emphasis on psychosomatic approaches to explain many symptomatic manifestations in patients can be dangerous. The following case is reported to alert radiologists against placidly accepting referring diagnoses which imply a lack of underlying organic foundation.

The case presented is interesting from the standpoint that the diagnosis over a period of five years was that of anorexia nervosa, that the patient maintained a keen insight into her illness, and that finally advanced lesions of her pancreas and cerebellum were recognized.

REPORT OF A CASE

History of Illness. M. M., a twenty-eight year old single female, college graduate, employed as a copywriter, entered this hospital on May 17, 1955. The chief complaints were hiccups, 15 pound weight loss, recent epigastric distress, frequent morning emesis, and anorexia for at least six years. The patient emphasized, "The hiccups are not a nervous manifestation. They even occur during sleep. I believe the hiccups are the cause of anorexia nervosa and not vice versa." Her family and menstrual histories were negative.

Earlier this year she noted increasing sensations of dizziness associated with blurring of vision and loss of balance, but she had never had a convulsion or lost consciousness.

Physical Findings. The patient was thin (93 pounds and 5 feet 8 inches tall) and apprehensive. A mild pectus excavatum deformity of the chest existed (Fig. 1). The blood pressure was 100/60, pulse 84, temperature and respirations normal. The abdomen showed a visible bulge in the left upper quadrant coinciding with a firm, slightly irregular and tender mass extending three and one-half finger breadths below the left costal margin. The fundoscopic examination disclosed indistinctness of disk margins, but no pathology was diagnosed by a consultant.

Laboratory Findings. The hematologic and chemical laboratory findings were all normal, and included hematocrit 33, white blood cell count 7,560, sedimentation rare 6, negative urinalysis, basal metabolic rate minus 8 per cent, alkaline phosphatase 1.0, thymol turbidity 1.7, and negative cephalin flocculation test after 48 hours. Other values were: potassium 4.9, sodium 140, nonprotein nitrogen 29, fasting blood sugar 95, chlorides 107, albumin 5, globulin 4.2, and cholesterol 186. Stools were negative for parasites.

Roentgen Findings and Roentgen Diagnostic Impression. Barium meal study on May 18, 1955, revealed displacement of the stomach anteriorly to the left and inferiorly by a probable pancreatic cyst (Fig. 3). Displacement of the duodenal loop with evidence of dilatation, which may suggest some encroachment upon the lumen distally, was noted. Chest roentgenograms showed the pectus excavatum deformity (Fig. 1). The intravenous pyelographic study was normal.

Surgical Findings. On May 25, 1955, the patient was operated upon and an enlarged multicystic pancreas was found. A subtotal pancreatectomy with a splenectomy was performed (Fig. 6). The fluid from the cyst was clear. The serum amylase test from the fluid was 50 units. Postoperatively, there was some wound drainage which was checked for tryptic activity, but was negative. Stools were negative for fat.

Postoperative Course. The patient was transferred back to the medical service because of hiccups, anorexia and occasional episodes of dizziness. There was no response to thorazine. A five hour glucose tolerance test was done; the fasting blood glucose was 132 mg. per cent, one hour later 330 mg. per cent, four hours later 228 mg. per cent, and five hours later 171 mg. per cent. This was interpreted as a diabetic curve due to the loss of pancreatic tissue from a previous operation. The prothrombin time was 67.5 per cent (normal 85–100 per cent). Potassium was 5.9 and sodium 143. The radio-

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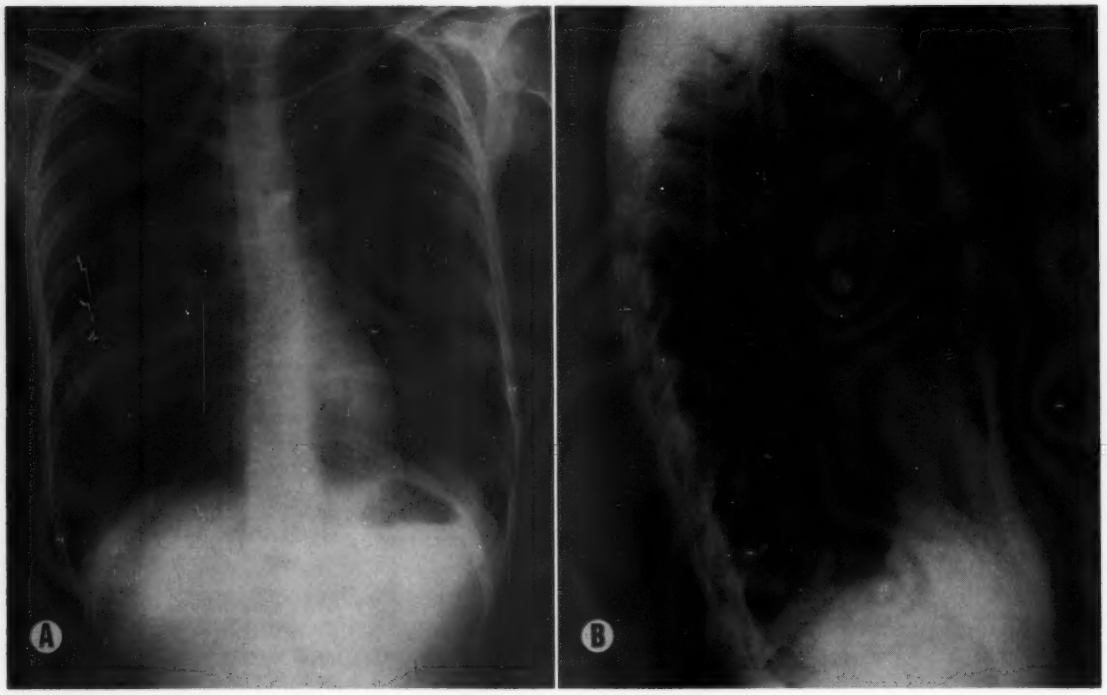

Fig. 1. (*A* and *B*) Pectus excavatum deformity of chest. Right lower hilus is prominent due to displacement of heart to left. No evidence of pulmonary fibrosis. Note extreme absence of subcutaneous tissue.

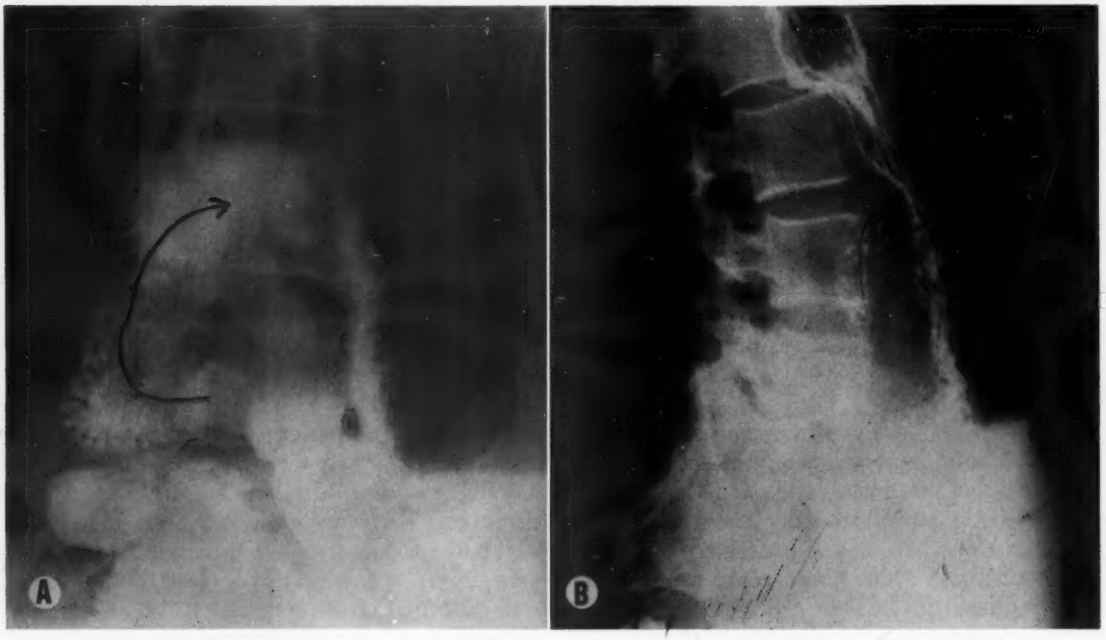

Fig. 2. (*A* and *B*) Duodenum inversum, or looped duodenum. See discussion in report.

Fig. 2. (*C*) The left lateral view of the stomach in 1952. See also Figure 2*B* and compare with Figure 3*B*.

active iodine uptake was 19.3 per cent, being within normal limits. The 24 hour 17 ketosteroids revealed 9.5 mg. (normal female value).

Neurosurgical Findings. After ventriculographic studies (Fig. 4), the patient was explored by way of a suboccipital craniotomy. A large anomalous blood vessel crossing over the vermis and into the fourth ventricle terminated in a mass of closely interwoven blood vessels filling the fourth ventricle. On both sides of this mass were large cysts which had herniated the tonsils through the foramen magnum. There was a great deal of bleeding during attempted resection and the patient was given 2,000 cc. of blood. Postoperatively, a tracheotomy tube and respirator were used, but the patient never did regain consciousness, and died six days later.

Autopsy Summary. Autopsy study revealed a large vascular mass in the region of the brain stem and cerebellum, softening of the medullary region of the brain, and adrenal rests of the kidneys. A small retroperitoneal abscess in the left hypochondriac region was noted. Microscopic study of the vascular tumor in the region of the fourth ventricle revealed a hemangioblastoma. With the prior surgical specimen of polycystic

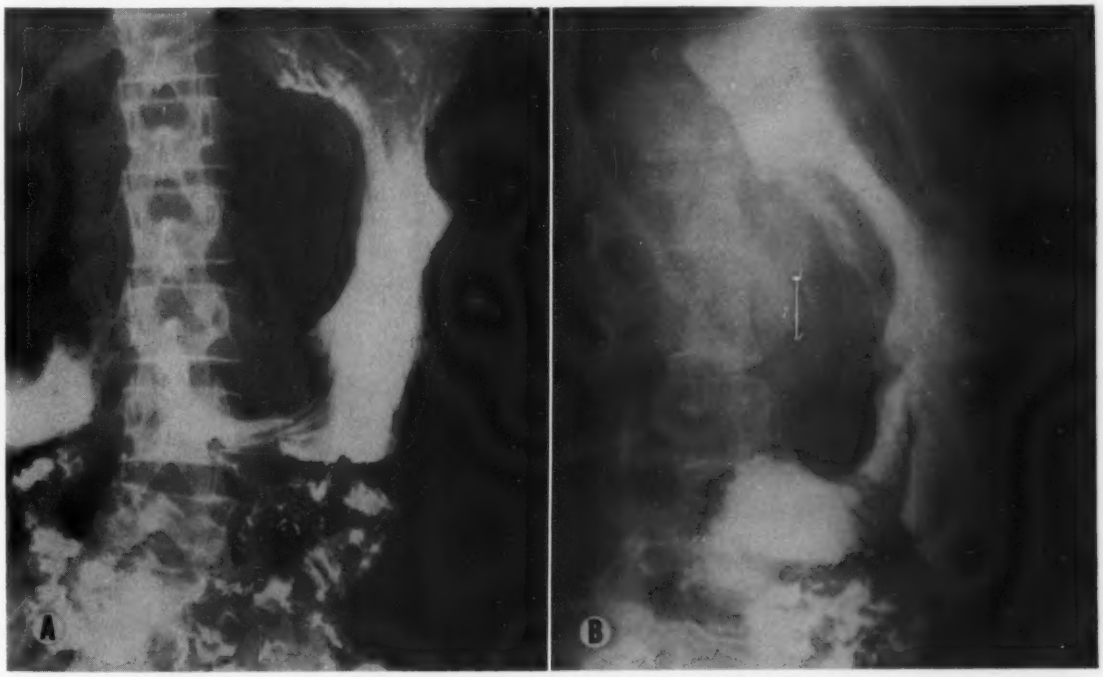

Fig. 3. (*A*) Posteroanterior and (*B*) left lateral roentgenograms of the stomach in August, 1955. See discussion and compare with Figure 2.

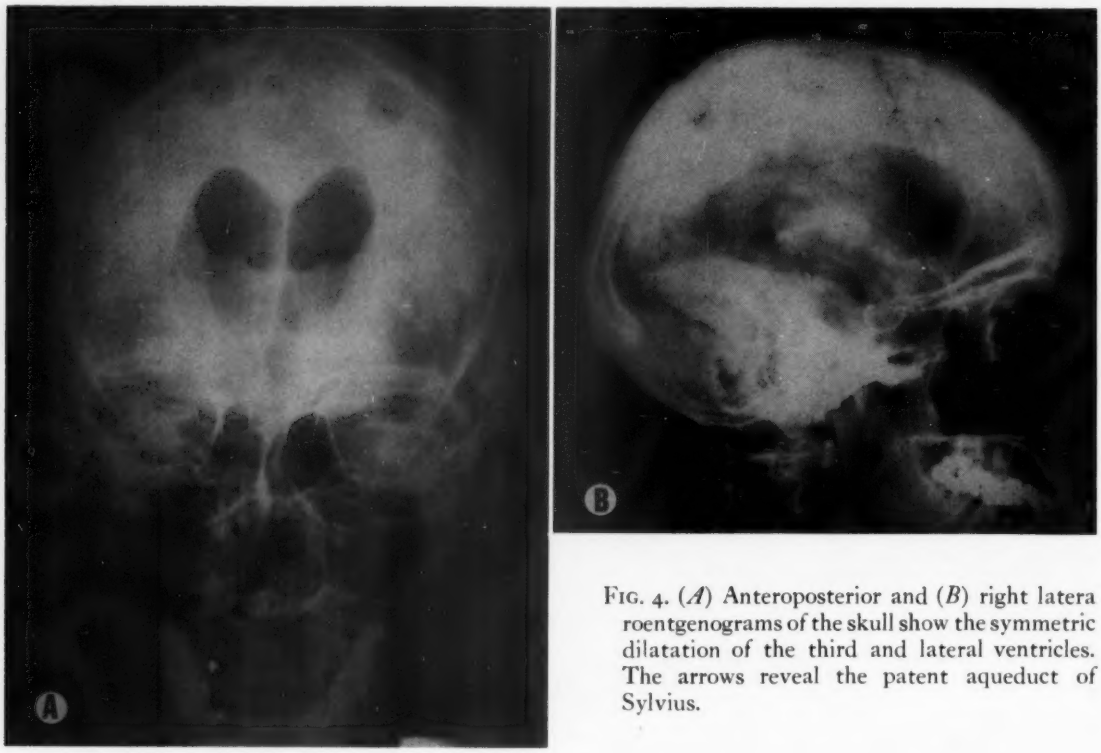

FIG. 4. (*A*) Anteroposterior and (*B*) right latera roentgenograms of the skull show the symmetric dilatation of the third and lateral ventricles. The arrows reveal the patent aqueduct of Sylvius.

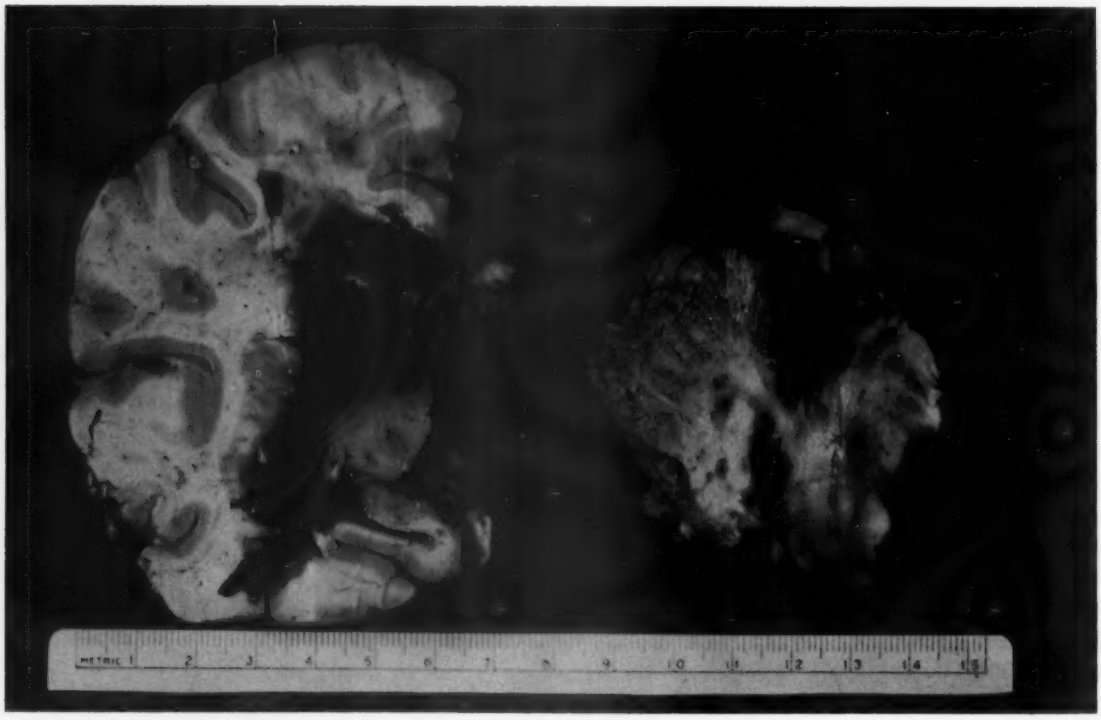

FIG. 5. Coronal section through the occipital region and a sagittal section through a portion of the brain stem. Cystic hemorrhagic areas due to the malformation and surgical interference.

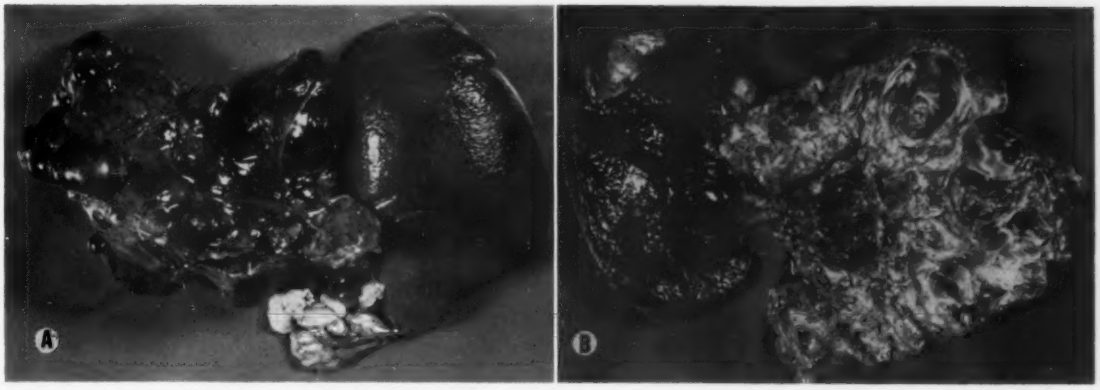

FIG. 6. Surgical specimen removed at first operation. Three-fifths of the polycystic pancreas and spleen. (*A*) Closed and (*B*) open views.

disease of the pancreas, Lindau's complex was verified.

DISCUSSION

The 1952 roentgen examination performed elsewhere was reported as showing a rather marked and prolonged reverse peristalsis in the second and third portions of the duodenum, with the point of delay appearing to be at the ligament of Treitz. The films revealed a mobile duodenum inversum, or looped duodenum (Fig. 2). This was attributed to redundancy of the second portion of the duodenum and may be associated with abdominal ptosis. This anomaly is usually devoid of any clinical significance in itself. Similar illustrated cases are shown in the basic texts of Shanks and Kerley,[12] Templeton,[14] and Buckstein.[3] The lateral roentgenogram of the stomach was not remarkable (Fig. 2*C*). There was nothing to indicate a primary pancreatic fibrosis with secondary lung changes. The chest roentgenograms showed the pectus excavatum deformity (Fig. 1). The lower right hilus was prominent due to the displacement of the heart. During the subsequent three year interval, the pancreatic lesion progressed, so that it was readily demonstrable. The stomach was displaced laterally, anteriorly, and inferiorly (Fig. 3, compare with Fig. 2). At surgery three-fifths of the pancreas was removed with the spleen (Fig. 6). Pathologic study reported a "congenital polycystic pancreas with papilliferous adenomatous features and marked fibrosis and atrophy of exocrine pancreatic tissue."

It is interesting that there was no dysfunction of the pancreas detected clinically. Postoperatively, abnormal amounts of fat were not found in the stool and only transient hyperglycemia was recorded on the glucose tolerance test, but insulin was never required at any time. This is to be contrasted with the report of Anfanger, *et al.*[1] of an emaciated nine year old boy who had pancreatic achylia and glycosuria due to congenital cystic disease.

Five cases of hemangioblastoma involving the fourth ventricle were reported by Walker and co-workers.[15] The patient's symptoms in greater part can be explained by the obstruction of the circulation of the cerebrospinal fluid at this level. The resulting symmetric hydrocephalus with dilated aqueduct of Sylvius is shown in Figure 4. Although our patient had a pancreatic lesion, the hiccups were most probably manifestations of cerebral involvement.[7]

It is known that the brain lesion can occur as an isolated vascular tumor.[9,13] The early historic notes concerning cystic hemangioblastoma and the surgical survival rates given by Perlmutter, *et al.*[10] are interesting. Noran's[8] recent revised classification and Bailey's explanation of the embryonic origin are helpful in understanding the disease process. Our patient's illness is best related to Lindau's disease.[6] Besides

the hemangioblastoma of the brain stem and polycystic pancreas, adrenal rests were found in the kidneys. Lindau has mentioned that hypernephromas of the kidney may be associated with the disease complex. The retinae were grossly normal. The immediate family failed to show any retinal changes. The von Hippel component was lacking, but Bailey mentions that it is necessary to have microscopic study to exclude it completely. Lindau[7] cites that only 20 per cent of cases have familial incidence and more often the angiomatous malformation of the cerebellum may occur as a solitary growth. In contrast to the present case is that of Isaac *et al.*[5] in which renal and pulmonary lesions were prominent.

SUMMARY AND CONCLUSION

A case of anorexia nervosa with associated lesions of the fourth ventricle and pancreas is presented. The roentgen findings and pathologic features are discussed.

The age, presence of angioblastoma with cerebellar cysts and congenital polycystic pancreas, together with adrenal rests of the kidney, identify the complex as Lindau's disease.

The marked pancreatic involvement without early clinical manifestations of dysfunction suggests the necessity of careful roentgenologic examination.

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Vol. 78, No. 2

RENAL BACKFLOW IN EXCRETORY UROGRAPHY

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RENAL backflow in pyelography refers to the escape of the contrast medium from the pelvis and calyces into the surrounding renal structures. Gigon in 1856, while preparing injection specimens of kidneys, first noted the escape of the injected material from the renal pelvis into the renal vein. Other investigators during the nineteenth century made similar observations, but the phenomenon attracted little attention until the introduction of retrograde pyelography by Völcker and Lichtenberg in 1906. Between 1906 and 1924 numerous authors reported cases of backflow during the course of retrograde pyelography and speculated upon the mechanism of the phenomenon. The majority of these authors considered the most likely route of extravasation to be via the tubules with rupture into the vessels.

In 1924 Hinman and Lee-Brown,[3] in making corrosion preparations of the kidney, found that in spite of great caution the material injected tended to flow into the vascular system of the kidney. They also demonstrated in studies carried out on cadaver specimens of mammalian kidneys and on living sheep, rabbits, and dogs that under moderate pressure, often less than the secretion pressure of the kidney, they could cause the contents of the renal pelvis to pass out through the renal vein. They felt that the transfer took place in the fornix of the minor calyces, and was due to a structural condition at the base of the pyramids where a rich plexus of veins lie in close apposition to the deep sulci. To designate this phenomenon they coined the word "pyelovenous backflow," a term which has been used with varying connotation ever since.

In 1925 Fuchs,[2] working independently on human cadaver kidneys, demonstrated that fluid injected into the renal pelvis would in most cases stream into the renal veins through ruptures in the fornix of the calyces. He concluded that backflow originated in the fornix by a rupture at the angle of maximal and minimal extensibility and that the extravasate then spread into the sinus renalis and the perivascular spaces. In this location the contrast medium pressed the vein away from the parenchyma thereby causing a rupture in the vascular wall through which the medium could stream into the veins.

Far reaching clinical and pathophysiologic conclusions were drawn from this experimental work without the support of clinical observations. Many reports of backflow appeared in the literature. For the most part these were case reports and descriptions of an occasional accident with retrograde pyelography. While the phenomenon was described as pyelovenous backflow, very few of the cases demonstrated any of the contrast substance in the renal vein. It was not until 1948 that Olsson[11] questioned the conclusions drawn by the earlier writers and investigated the problem from both the clinical and experimental viewpoint. In 1953 Köhler[6] also investigated the mechanism of backflow from the clinical as well as the experimental viewpoint and agreed with the opinion of Olsson.

The work of both Olsson and Köhler tended to prove that the most common type of backflow consists of a small extravasation of contrast medium through a fornix rupture into the renal sinus, a structure consisting of adipose and connective tissue which supports a venous plexus. In the roentgenogram this is manifested by a thorn-like process extending from the fornix of a minor calyx into the kidney substance (Fig. 1 and 2). When the extravasation is more extensive, it spreads perivascularly

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along the hilus and retroperitoneally downward along the ureter (Fig. 3 and 4). In occasional cases the extravasate enters the lymphatics. In the roentgenogram this is manifested by several fine linear streaks 1 to 2 mm. wide which extend from the sinus extravasate horizontally toward the vertebral column (Fig. 5). Only rarely the extravasate actually penetrates the renal vein and enters the general circulation. In proof of the latter both Olsson and Köhler showed that first, the contrast medium was almost never seen in the renal vein. Second, both added fluorescein sodium to the contrast media used in retrograde studies.

Fig. 1. Excretory urogram, film exposed 15 minutes after injection of neo-iopax. No significant urinary symptoms. Thorn-like process extending from fornix of upper lateral minor calyx. Early spread of contrast medium into sinus renalis about inferior minor calyx and also superior lateral minor calyx.

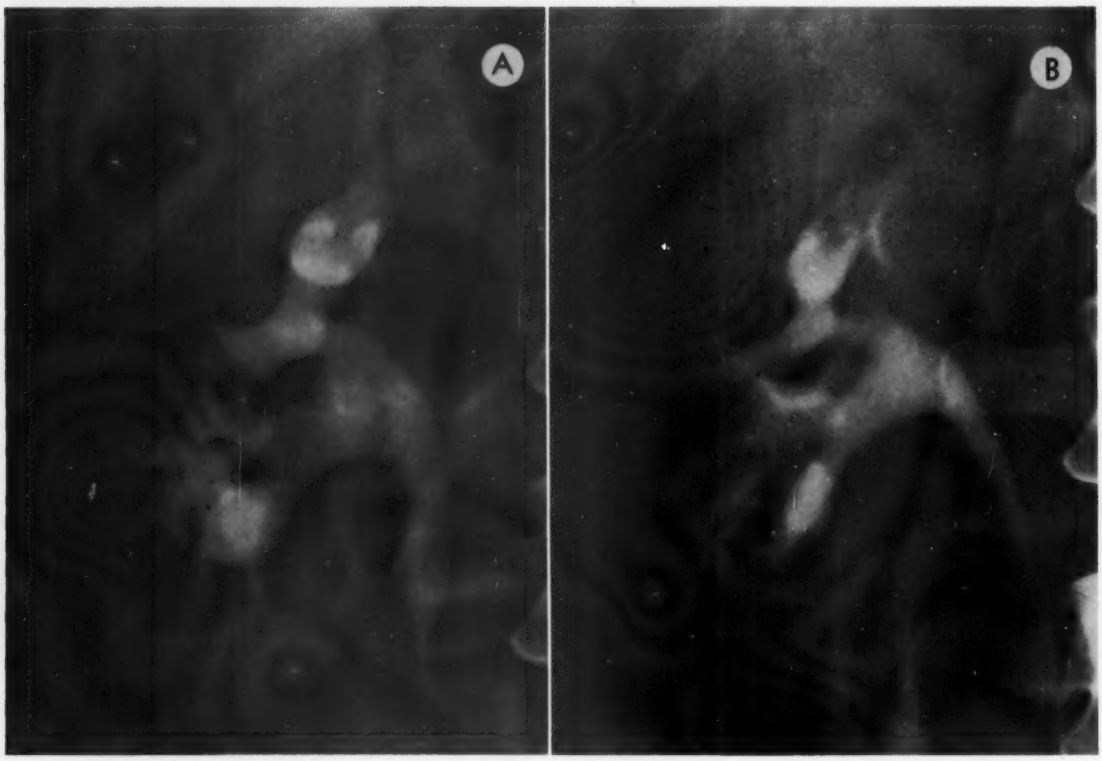

Fig. 2. Excretory urogram performed to investigate a questionable right upper quadrant mass. No urinary symptoms. (*A*) Film exposed 10 minutes following injection of neo-iopax. Early sinus extravasate from fornix of middle minor calyx. (*B*) Film exposed 15 minutes following injection. Contrast medium has spread into sinus renalis about middle calyx. Also note early sinus extravasate from fornix of superior minor calyx.

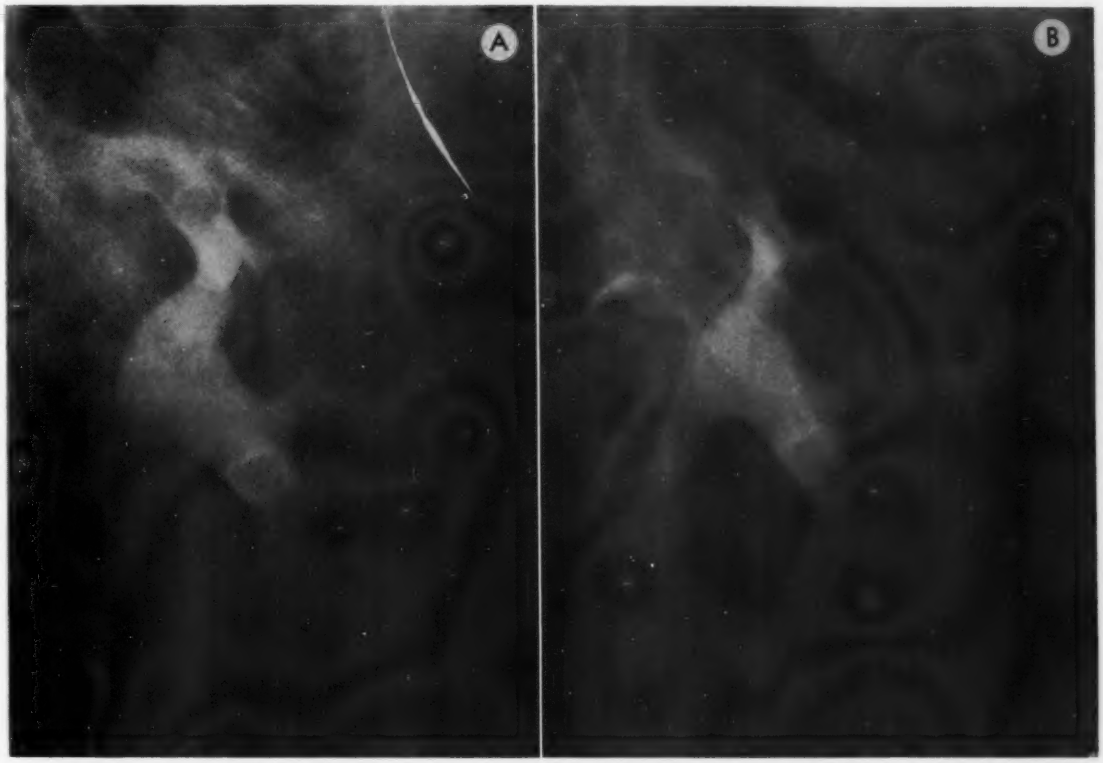

Fig. 3. Excretory urogram performed in routine preoperative work-up of patient with carcinoma of rectum. No urinary symptoms. (*A*) Film exposed 5 minutes following injection of neo-iopax. Very small sinus extravasate from medial fornix of superior minor calyx. (*B*) Film exposed 15 minutes after injection. Extensive spread of the contrast medium perivascularly along the hilus and retroperitoneally downward along the ureter.

This material causes fluorescence of the skin and mucous membranes in very dilute quantities. Only in an occasional case where extensive extravasation had occurred could the material be demonstrated in the general circulation.

In pyelotubular backflow the mechanism of extravasation differs from that seen in fornix rupture. In the roentgenogram, pyelotubular backflow is manifested by wedge-shaped opacities having a somewhat brush-like appearance which arise from the middle of the calyx rather than from the fornix and widen out in a cortical direction (Fig. 6). Usually, the opacities are small but they could extend peripherally as far as the renal capsule. They always maintain their wedge shape. The backflow probably occurs through the openings in the renal papilla and into the distal tubules.

Many reports describing backflow in retrograde pyelography have appeared in the literature. In sharp contrast only two or three reports of the phenomenon occurring in excretory urography can be found in the English literature. Olsson credited Fuchs (1931) with first mentioning backflow in a patient who had excretory urography done in association with an attack of renal colic. In 1938 in a discussion of renal tuberculosis, Narath pointed out an example of pyelolymphatic backflow during the course of an intravenous pyelography. In 1943 Lindbom[8] and Steinert published the first detailed report of backflow in excretory urography based upon 15 cases of fornix backflow. In 1948 Olsson published his monograph on "Studies on Backflow in Excretory Urography."[11] In this excellent work he analyzed 100 intravenous pyelograms exhibiting backflow, and correlated his findings with previous experimental work in retrograde studies. The paucity of material on this subject in the

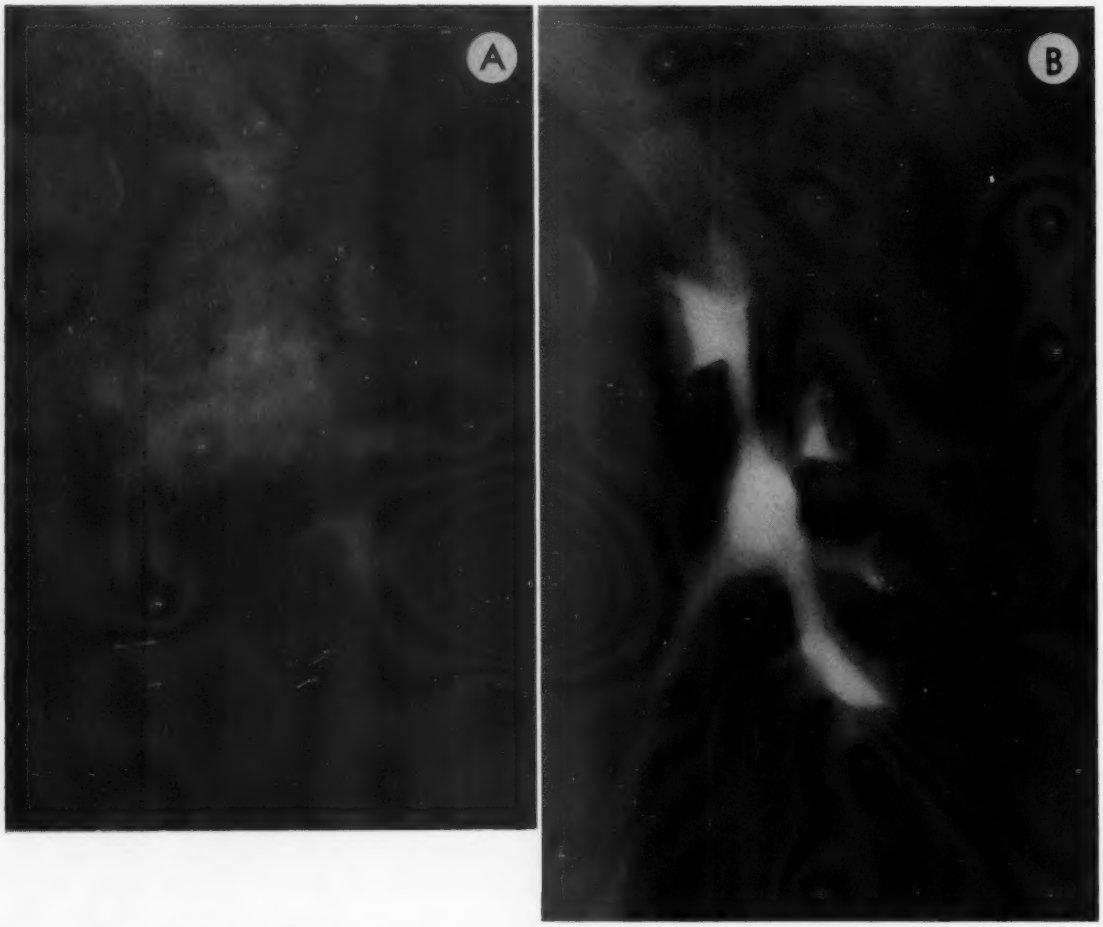

Fig. 4. (*A*) Excretory urogram made because of pain in left back. Urine negative. Film exposed 5 minutes after injection of neo-iopax. Extensive sinus extravasation perivascularly along the hilus and retroperitoneally downward along the ureter. (*B*) Retrograde pyelogram taken the following day. Normal renal architecture.

American literature prompted this report.

The incidence of backflow in excretory urography is mentioned in two reports. Lindbom in a series of 900 cases in which ureteral compression was employed found 15 examples of backflow, an incidence of 1.6 per cent. Olsson in a series of 988 cases in which ureteral compression was employed found 37 cases exhibiting backflow, an incidence of 3.7 per cent. No attempt is made in the present report to establish the incidence of the phenomenon, but surely it is not an unusual occurrence during routine pyelography.

Several illustrative examples are included to demonstrate the various types of backflow. In these cases the roentgenographic technique employed consisted of the usual preparation of witholding fluids for a least 12 hours prior to the examination and the administration of a mild laxative the night before. After sensitivity tests the contrast medium (neo-iopax or hypaque sodium) was injected intravenously. Ureteral compression was obtained by the use of a blood pressure cuff placed on the lower abdomen above the symphysis pubis and under a restraining band. Five minutes after the injection was completed, the cuff was inflated to between 110 and 120 mm. mercury pressure. Films were exposed 10 and 15 minutes following completion of the injec-

tion. The cuff was deflated and another film exposed 20 minutes following the injection. If these urograms showed nothing unusual, a film in the upright position was exposed 30 minutes following the injection. Ureteral compression was used in all cases where it could be tolerated by the patient and was not contraindicated by some abdominal disease.

Obstruction to the ureter resulting in increased intrapelvic pressure represents the one necessary requirement for the possible development of renal backflow. Most commonly, the obstruction is caused by the compression device employed during roentgenography, and in these instances, the backflow may be considered as a roentgen artefact. In recent years the use of a blood pressure cuff or similar rubber ballon under a restraining band has resulted in more effective ureteral compression. These devices produce a substantial increase in intrapelvic pressure, and in the susceptible individual the potential for backflow exists frequently. In Olsson's series of 100 cases, 94 occurred after the application of ureteral compression. Of these he classified 9 as pyelolymphatic, 2 as possibly pyelovenous, and 83 as sinus extravasates. Sex distribution in his series was equal but the phenomenon was found to occur much more frequently in patients under fifty years of age than in the older age group suggesting that the tonus of the renal pelvis played some part in its development. The interval between application of compression and the appearance of backflow varied from 9 to 61 minutes averaging 20 minutes.

Fig. 5. Retrograde pyelogram. Characteristic fine linear horizontal streaks of contrast medium in pyelolymphatic backflow.

Fig. 6. Excretory urogram. Film exposed 20 minutes following injection of hypaque sodium. Wedge-shaped opacities arising from the middle of the fornices in pyelotubular backflow or tubular stasis.

Acute renal colic is caused pathophysiologically by an increase in intrapelvic pressure due to obstruction of the ureter usually by a calculus. This increase in intrapelvic pressure may also result in backflow. The roentgenogram in these cases is characterized by the very early appearance of the extravasate usually on the first exposure following injection and by the exten-

Fig. 7. Excretory urogram performed 4 hours after onset of acute right renal colic. Small calculus demonstrated in pelvic ureter. Film exposed 60 minutes after injection of hypaque sodium. Extensive sinus extravasate extending perivascularly along hilus and downward along ureter. Calculus recovered 48 hours later.

sive extravasation. The contrast substance can be seen arising from the region of the fornices of the calyces from where it spreads to the renal sinus and around the hilus vessels and downward along the ureter (Fig. 7 and 8). The prompt appearance of the backflow in these cases suggests the probability that a backflow passage had already been forced prior to injection of the contrast medium. Six of Olsson's 100 cases fell into this category.

COMMENT

The significance of renal backflow occurring following the application of ureteral compression is to recognize the phenomenon as a urographic artefact and not to ascribe it to any important pathologic process. It should not be confused with traumatic lesions of the kidney or tuberculosis where the differential diagnosis is quite obvious. Backflow visualized during an attack of renal colic offers positive and dramatic evidence of stasis and ureteral obstruction. Its roentgen appearance is quite characteristic and should not be confused with that of tuberculosis. It is important to remember that the presence of rather extensive extravasation does not indicate perinephritis or seepage into a perinephritic abscess. Renal backflow has not been reported where ureteral compression has not been employed or where no obstruction of the ureter existed. In excretory urography, especially

Fig. 8. Excretory urogram made 6 days following onset of left renal colic. Film exposed 15 minutes following injection of neo-iopax. Small calculus overlying transverse process of third lumbar vertebra. Extensive sinus extravasation spreading perivascularly along hilus and retroperitoneally downward along ureter. Emergency operation 3 hours later revealed no rupture of ureter or pelvis and no evidence of extravasated urine in the perirenal or periureteral areas.

with the newer, more concentrated contrast media, the picture of pyelotubular backflow is probably the result of tubular stasis rather than actual extravasation of the medium into the papilla.

SUMMARY

1. The American literature contains few references to backflow in excretory urography.

2. The term "pyelovenous backflow" does not accurately describe the extravasations seen during urography.

3. Sinus extravasations are the most common type of backflow.

4. Renal backflow seen following the application of ureteral compression is essentially a urographic artefact.

5. Extravasation demonstrated during acute renal colic affords positive evidence of stasis and increased intrapelvic pressure.

6. Illustrations of renal backflow are presented.

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PYELORENAL BACKFLOW*

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ROUTINE work in diagnostic roentgenology offers an opportunity for observation of unexplained phenomena of incompletely determined significance. Of such, the appearance of backflow in retrograde and excretory urography is an example. Backflow, or the escape of opaque material from the calyces, is suggested by the appearance of opaque medium in lymphatics or elsewhere. The present study grew out of the observation of backflow in routine clinical material. It is hoped that this report may be of possible help in the eventual, complete determination of the significance of backflow.

FIG. 1. Bulb tip catheter. Bulb tip catheter in left ureter, already removed from right. From the pelvis of the right kidney, a few thin lines of opacified lymphatic vessels can be seen. From the left kidney the lymphatic filling is more clear cut. In the left kidney substance, cortical interstitial streaking is noted. Some advanced pyelosinal backflow is present.

If the opaque medium is visualized within the collecting tubules, the backflow is pyelotubular (Fig. 6, 7, 8, 9, 10 and 11). Sometimes, the lymphatic vessels from the kidney leading to the para-aortic lymph nodes are opacified by the urographic medium. This is then referred to as pyelolymphatic backflow (Fig. 1, 3, 4 and 5). The occurrence of pyelovenous backflow has been demonstrated in cadaver specimens and suggested in roentgenograms from living patients. In pyelovenous backflow the opaque medium is seen in a position which outlines the pattern of venous drainage within the kidney (arcuate veins, etc.). Also, there are patterns of pyelorenal backflow which seem to indicate that the opaque medium has penetrated interstitially or is within the sinus renalis. Direct rupture of a

* This work was undertaken at the suggestion and under the supervision and guidance of Dr. Harold Peterson who made available the facilities of his X-Ray Department at Miller Hospital, St. Paul. Thanks are due also to Dr. Kano Ikeda, Pathologist, who made possible the experimental work on cadaver specimens.

Fig. 2. Regular catheter. Diffuse interstitial extravasation has developed in streaks and fan-shaped patterns in upper pole and medially at and below the ureteropelvic junction.

Fig. 3. Regular catheter. Lymphatic filling is now visualized leading from the upper part of the renal pelvis medially upward toward the spine. This kidney is ptotic with its upper pole at L-3; otherwise, the lymphatic pathway would probably be inclined slightly downward.

Fig. 4. Regular catheter. Pyelolymphatic backflow.

calyx during retrograde pyelography would result in pyelointerstitial backflow (Fig. 1, 2, 5, 8, 10 and 11).

Pyelosinal backflow provides a subtle differentiation. Characteristically there is, between the extravasate and the medium within the calyx, a space of about 1 mm. which corresponds to the thickness of the calyceal wall (Fig. 1, 6, 7, 9, 10 and 11).

The sinus renalis constitutes an extension of the hilus into the kidney substance. It is a potential space with areolar tissue, nerves, lymphatics and blood vessels. It is lined by a tunic of fibrous tissue extending from the surface of the kidney at the hilus. This potential space becomes outlined by the contrast substance probably as a result of fornix rupture which permits escape of the contrast medium into the sinus. Spread then occurs along the course of the vessels leading from the sinus toward the hilus. This causes horn-shaped extravasates, projecting always from the calyceal borders.

CLINICAL PRODUCTION OF BACKFLOW

Pyelorenal backflow has been observed in excretory as well as retrograde urography. Olsson[2] describes extensive backflow in patients with acute renal colic, who were

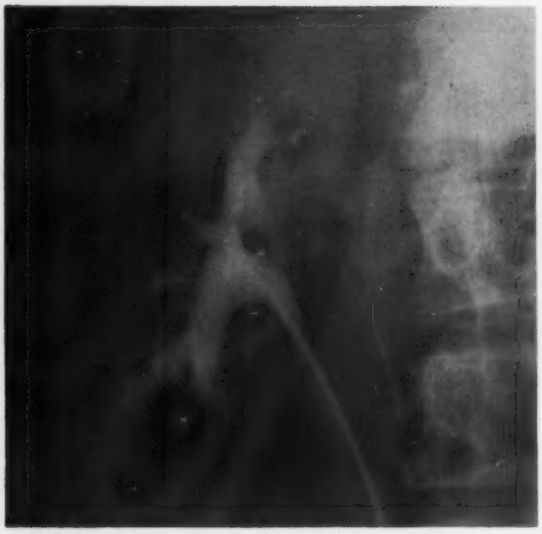


Fig. 5. Regular catheter. Pyelolymphatic backflow. The lymphatic vessels run medially at first and then turn downward in a narrow opaque line.

examined by excretory urography. Acute colic is productive of increased intrapelvic pressure as a result of spasm or obstruction to the discharge of urine. Sometimes this increase in pressure results in delayed or suspended excretion of urine and, therefore, of contrast substance. Sometimes urine excretion is evident but the increased pressure results in extravasation. In 6 cases, Olsson demonstrated such extravasation by excretory urography performed during an acute attack of pains and despite no preparation of the patient. In 4 of 6 cases calculi were found in the pelvic portion of the ureter. Excretion was delayed but finally the opaque medium revealed dilatation of the renal pelvis and ureter and a continuous opaque filling down to the stone. Dilatation was not of a high degree. A contrast extravasate developed, denoting rupture of a fornix. The extravasate spread in the renal sinus and into the retroperitoneal space down along and around the ureter. In all cases the extravasate was visible a relatively short time after the beginning of excretion of the contrast medium, suggesting es-

Fig. 6. Regular catheter. Pyelosinal backflow is suggested in the uppermost calyx where a narrow radiolucent zone is seen between the cortex and the extravasate.

Fig. 7. Bulb tip catheter. Right ureter only has been injected (compare with Figure 1). Pyelosinal backflow at an advanced stage is seen on the underside of the superior calyx and is suggested at the lower calyces. Streaking interstitial filling suggests contrast medium in collecting tubules.

FIG. 8. Regular catheters. Pyelointerstitial backflow. Some filling at tips of calyces—possibly excretory tubes. Note also diffuse fan-shaped filling with contrast medium at tip of calyx just above middle of left kidney. This extends fully from calyx to capsule of kidney.

FIG. 9. Bulb tip catheter. The lower calyx shows pyelosinal backflow. Similar extravasates are suggested at other calyces except the upper middle calyx. Here the fan-shaped filling with contrast medium is seen again.

FIG. 10. Bulb tip catheter. Pyelointerstitial backflow and suggestive evidence of collecting tubule filling are here manifested.

FIG. 11. Regular catheters. Pyelosinal and collecting tubule filling and unclassified backflow from calyces to kidney capsule.

cape through a passage already existent prior to injection. Increased pressure produced by colic is believed to have caused the rupture. The contrast injection merely provided visual proof of the rupture.

Excretory urography often provides evidence of backflow as an artefact in patients without colic. This artefact depends upon the application of pressure over the ureters, usually at the brim of the bony pelvis. Such application of external pressure is common practice, intended to produce stasis in order to back up enough contrast medium-containing urine so as to give ideal visualization of the ureters, pelves, and calyces. When pressure is well applied following good preparation of the patient, the resultant roentgenograms equal or surpass in diagnostic value the films obtained by retrograde urographic study. The induced stasis produces increased intrapelvic pressure and may result in extravasation or backflow.

In retrograde urography the introduction of the bulb tip catheter has provided an improved method for the visualization of ureters. These catheters are wedged into the distal ureteral orifices and the opaque medium is injected. The result is excellent visualization of the ureters bilaterally, as well as of the pelves and calyces. Many examples of backflow have been found in urographic studies made with bulb tip catheters (Fig. 1, 7, 9 and 10). It is assumed that greater pressure is applied within the system when these catheters are employed; nevertheless, there are many illustrations of similar backflow when ordinary ureteral catheters are employed (Fig. 2, 3–6, 8 and 11). Frequently, the rule of thumb utilized by the examining physician is to stop injection when slight pain in the kidney region is experienced by the patient, but pain may not develop before enough pressure has been applied to cause backflow. There are differences in the frequency with which certain types of backflow occur in excretory and retrograde urography. In both cases backflow occurs when there is a build up of pressure in the system. With the bulb tip catheter, as with application of pressure over the ureters by balsa wood blocks or other means, a "closed system" is responsible for build up of pressure. In excretory urography this build up is gradual except in cases of colic. In retrograde urography, the pressure increase is frequently abrupt and certainly more rapid.

CLINICAL OBSERVATIONS

The author reviewed both excretory and retrograde urograms at the Miller Hospital. In 40 cases of retrograde pyelography

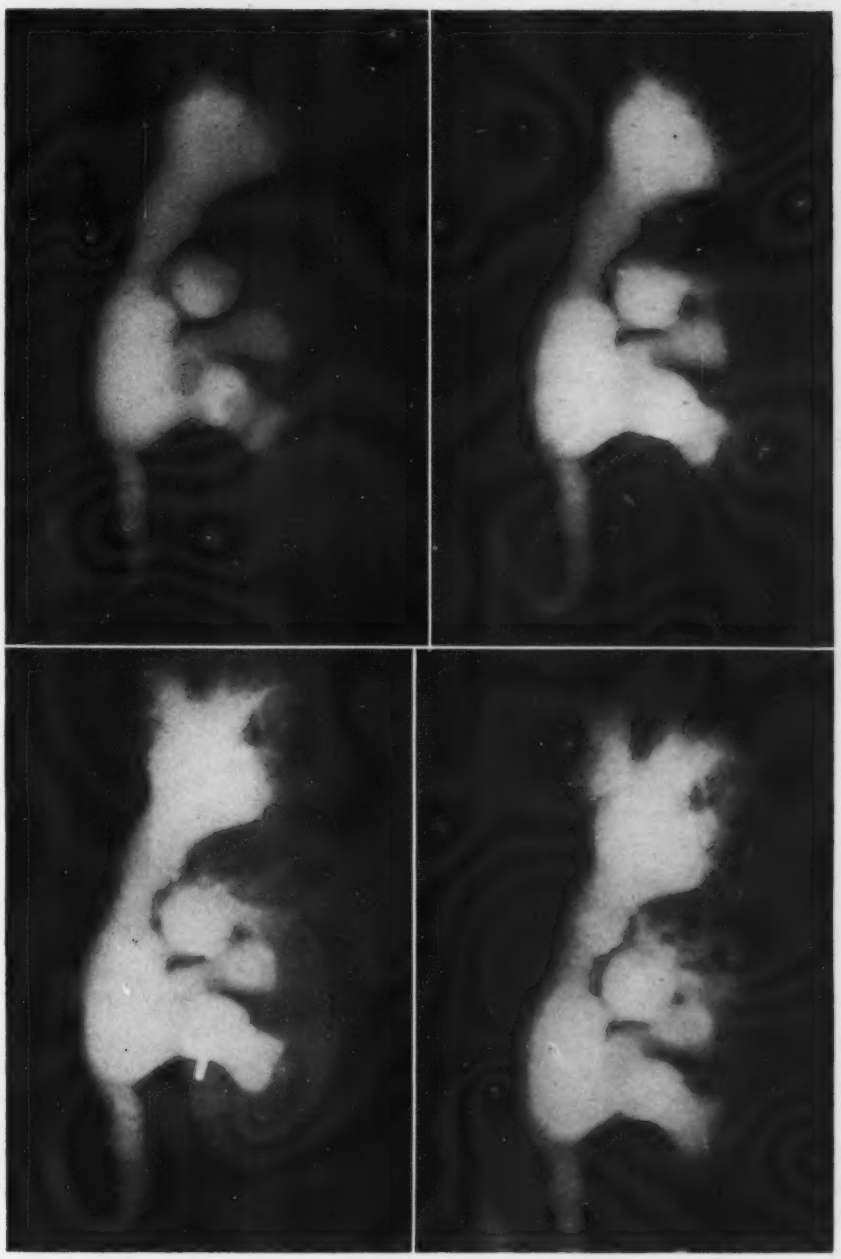


Fig. 12. Experiment 1. Ureteral injection with skiodan by gravity flow. *Upper left:* No backflow was evident on the first 5 films. *Upper right:* Collecting tubule filling is suggested. *Lower left and right:* Pyelosinal and pyelovenous backflow.

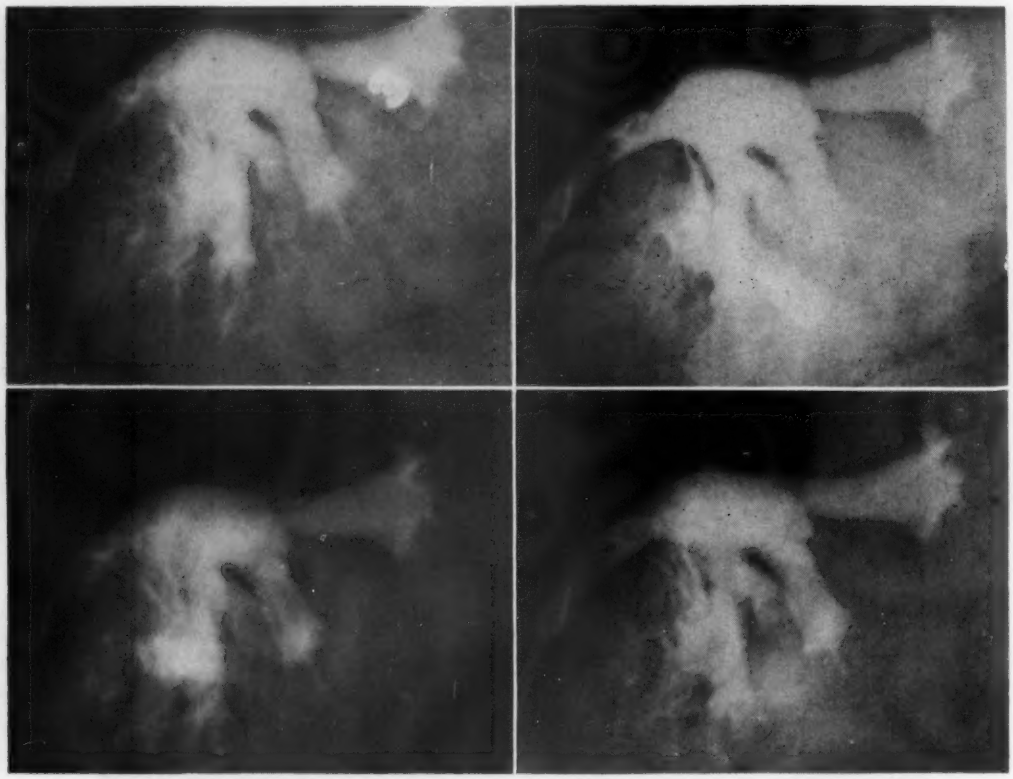


Fig. 13. Experiment II. Ureteral injection with skiodan by gravity flow. *Upper right:* Pyelosinal backflow from the lower calyx with spread to the hilus through the sinus renalis. *Upper left and both lower:* Spread into the lymphatics is suggested.

which showed backflow, the clinical data were carefully studied in order to determine any possible correlation between occurrence of backflow and the physical condition of the patient. Such matters as cardiovascular status and estimation of venous pressure were checked in the material on file in the record room and no correlation was noted. No relation to age, sex, or to the normalcy of the kidney was found.

The question has been raised whether pathologic conditions may be responsible for backflow. Some workers have contended that inflammatory processes in the kidney pelvis favor the occurrence of backflow, but this has not been shown in the present material.

The Miller Hospital roentgen film files provided 100 cases of pyelorenal backflow. Repeat occurrences were found. Examples of pyelotubular backflow were found on retrograde study in patients whose previous excretory study had shown pyelotubular backflow also. The material revealed a different type of backflow occurring on repeat studies. There was no definite indication that backflow, having been seen on retrograde or excretory examination once, was to be found on a repeat study of either type. Frequently, one roentgenogram showed several types of backflow.

These cases exhibited a much lower frequency of pyelolymphatic backflow on excretory urography than was found in the material of Olsson. The probable reason for this is that Olsson continued compression over a longer period of time. His initial application of compression was eight to fifteen minutes after injection and the appearance of backflow occurred from nine to six-

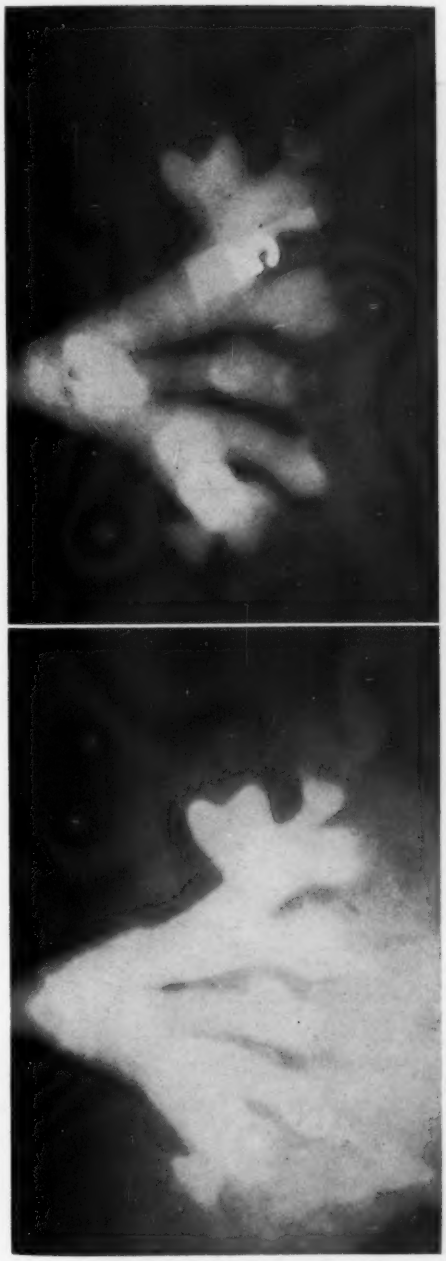


Fig. 14. Experiment III. Ureteral injection with skiodan by gravity flow. *Upper:* A horn-shaped extravasate is seen in the lowermost minor calyx of the superior major calyx following injection of 13 cc. of the contrast medium. *Lower:* The pyelosinal backflow is more developed with interstitial backflow and other pyelosinal extravasates in the kidney elsewhere.

ty-one minutes after compression with an average time of twenty minutes. In most routine studies at Miller Hospital, compression had been discontinued twenty minutes after it was applied.

The Miller Hospital material contains a higher percentage of cases of pyelolymphatic backflow in retrograde urography than is considered as average according to the literature. Various authors have denied that pyelolymphatic backflow is related to the injection pressure. Experience in the Miller Hospital series suggests a direct correlation. Several of the urologists practic-

Fig. 15. Experiment IV. Arterial injection by syringe with 50:50 lipiodol and mineral oil. The puzzling findings in clinical material and earlier experiments suggested the need for better understanding of the roentgenographic pattern of the vascular network. Faint outline of the pelvis and calyces is provided by 2 cc. of skiodan into the ureter.

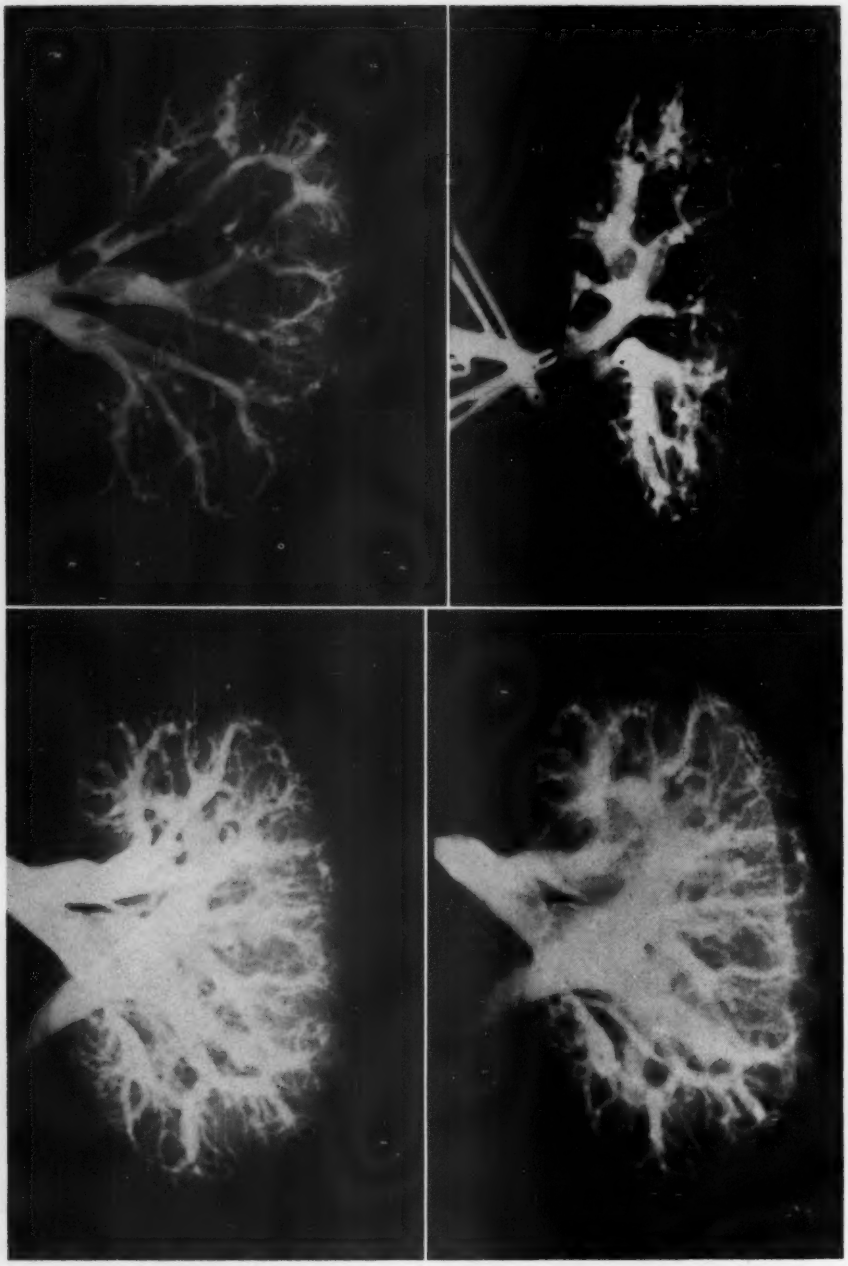


Fig. 16. Experiment vi. Venous injection by syringe with pantopaque. *Upper:* Anteroposterior and lateral views following injection of 4 cc. of pantopaque. *Lower:* Anteroposterior views following injection of 8 cc. of pantopaque in the vein and 5 cc. of skiodan in the ureter.

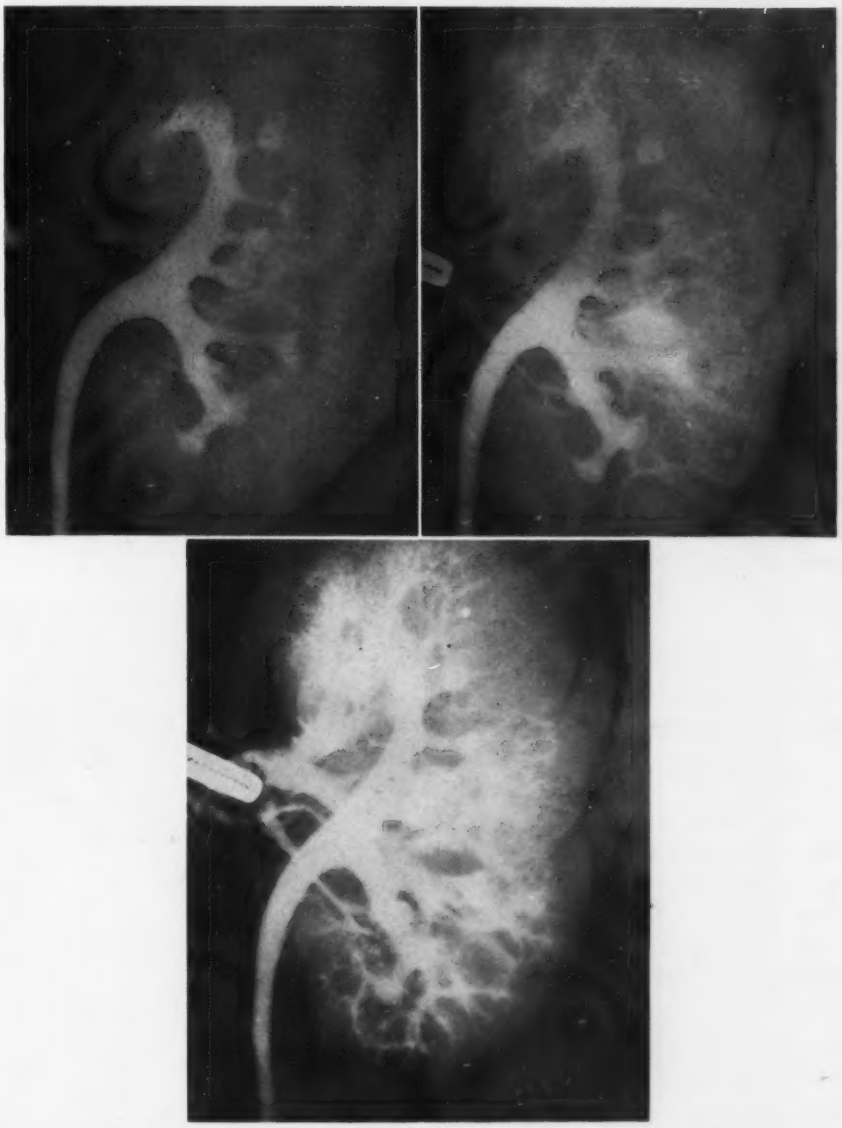


Fig. 17. Experiment v. Ureteral injection of skiodan by syringe. This is the opposite kidney to that shown in Figure 16. *Upper left:* Four cc. of skiodan by slow injection. Some leakage of contrast medium at the hilus was noted and a clamp applied as shown in the film to the right. Pyelosinal and pyelolymphatic backflow have developed. Pyelovenous backflow has begun. *Upper right:* Six cc. of contrast medium injected. Clamp is on the renal vein through which extravasated contrast material was escaping. *Lower:* The pattern resembles that in Figure 16 where contrast material was injected into both vein and ureter whereas here the medium was introduced into the ureter only.

ing in this hospital long ago adopted the use of the bulb tip catheter which gives a more complete filling and dilatation of the ureters, pelves, and calyces than is usually obtained. It may be that men who are accustomed to the use of the bulb tip catheter exert more pressure on injection with the ordinary catheter than they otherwise would. At any rate the percentage of pyelolymphatic backflow cases even with the ordinary ureteral catheter is high in this material (Fig. 3, 4 and 5).

Not all of the observed backflow can be classified easily as pyelolymphatic, pyelotubular, or pyelosinal or even pyelointerstitial. Pyelosinal and pyelointerstitial classifications must be broadened to include some of these cases. The illustrations reveal several types of backflow in a single film. Inability to explain or classify all the backflow patterns seen led to experiments on cadaver specimens in search for an explanation.

The clinical material chosen for illustration represents the problem cases. The uncomplicated material is purposely omitted.

AUTOPSY STUDIES

Olsson[2] and others reported studies in fresh kidney specimens from surgery and the morgue. Pyelosinal and pyelovenous backflow are readily obtained on such specimens. Olsson concluded that there is a decided difference between clinical and experimental backflow. He infers that little help can be obtained from autopsy injections for the interpretation of backflow under vital conditions. He and others do not report pyelolymphatic backflow in postmortem specimens. He seriously questions the occurrence of pyelovenous backflow in clinical material.

My experience is fortunately different in that I find the postmortem studies helpful for interpretation of the puzzling types of backflow in the clinical illustrations (Fig. 1, 2, 4, 6 and 8–11). Films were obtained to show the roentgenographic appearance of the arterial and venous patterns of the kidney vessels by direct injection of the renal artery and vein (Fig. 15, 16, 18, 23 and 24). Subsequent injections of the ureter, simulating retrograde pyelography, demonstrated several types of backflow. Pyelovenous backflow was most readily obtainable, as observed by Olsson. I gathered from his remarks that other types of backflow might not be demonstrable because the opaque medium escaped into the veins too readily.

The idea occurred that it might be possible to prevent pyelovenous backflow in the cadaver specimens by filling the veins with blood. (Olsson and others used air or water.) Fresh kidneys were obtained at autopsy. Care was taken to include as much of the perirenal fat as was possible with the kidneys. The blood vessels were clamped off. Out-dated blood from the blood bank was obtained and injected into the arteries

Fig. 18. Experiment VI. Arterial injection of 7 cc. of pantopaque shows free communication of renal arterial branches with perirenal fat vessels.

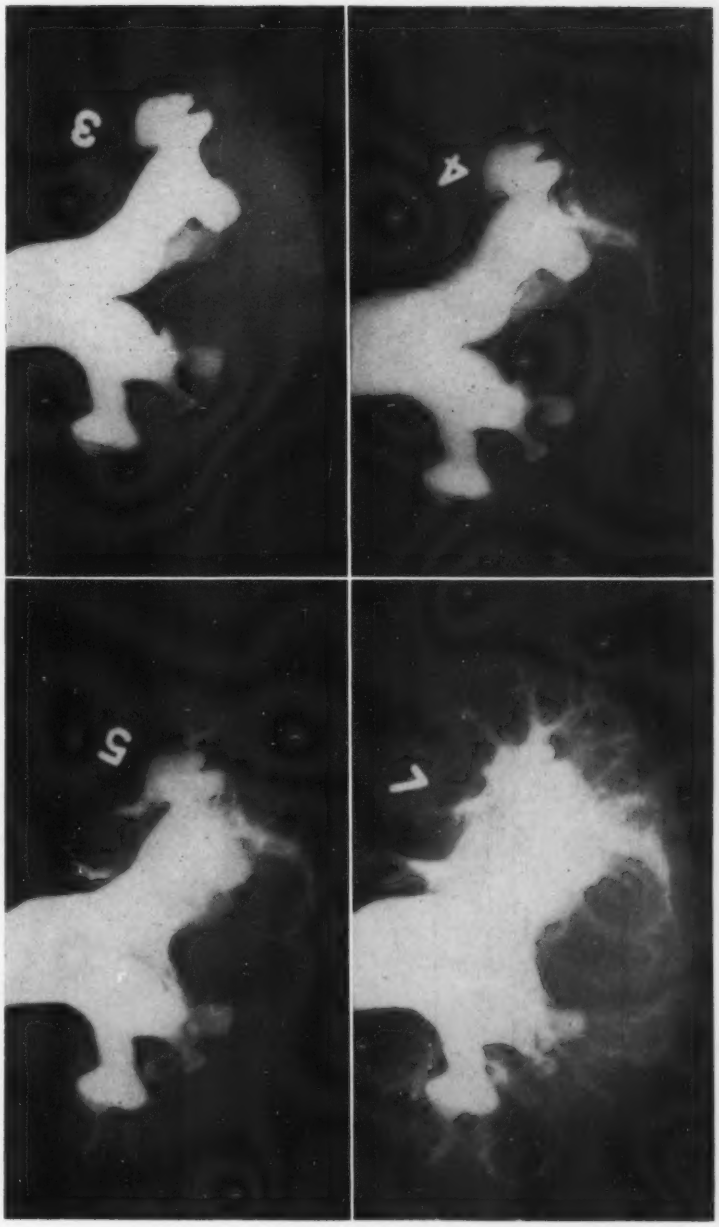


Fig. 19. Experiment VII. Ureteral injection with 50 per cent neo-iopax. *Upper left:* Pyelosinal backflow after injection of 12 cc. of medium. *Upper right and lower left:* Pyelosinal, pyelointerstitial, and pyelovenous backflow with the start of pyelolymphatic backflow after 14 cc. of medium. Note the persistence of a thin radiolucent line between the contrast medium in the sinus renalis and the contrast medium within the pelvis at the angle between the major calyces. *Lower right:* Pyelolymphatic backflow in later phases.

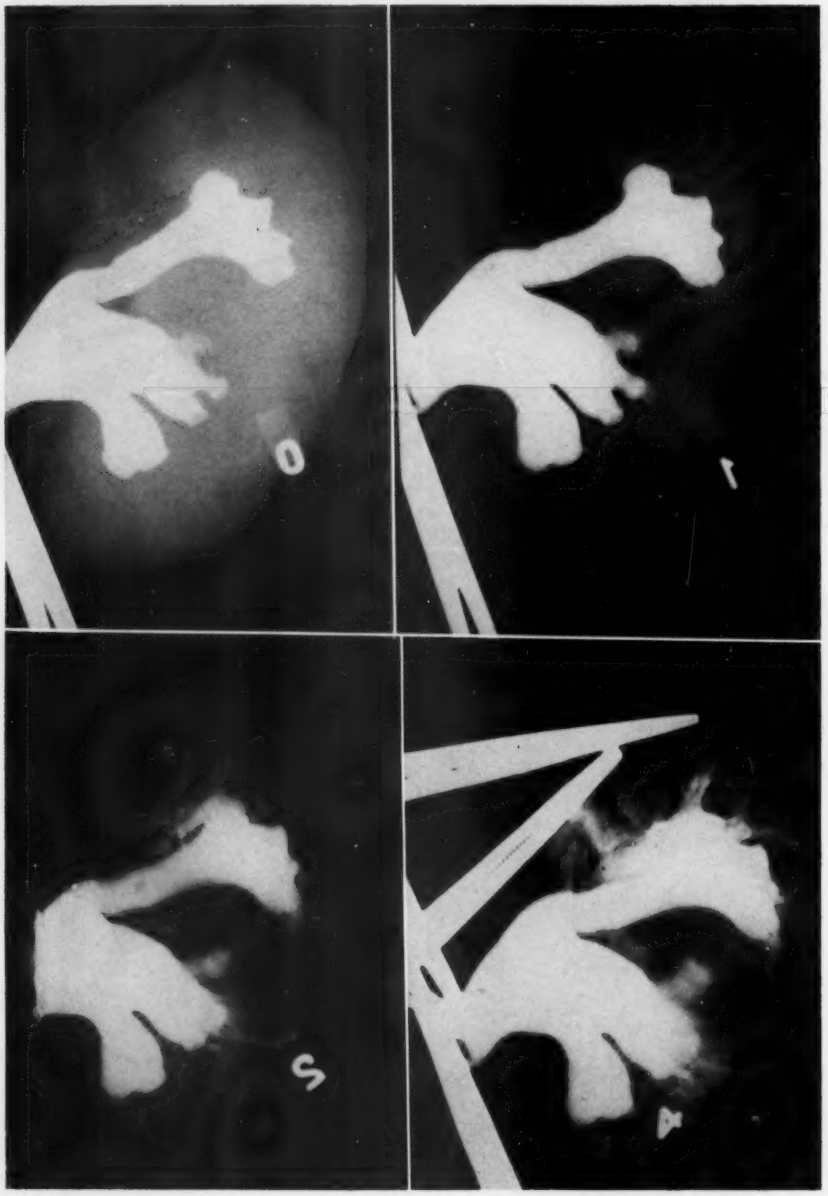


Fig. 20. Experiment vii. Ureteral injection by neo-iopax of the opposite kidney to that in Figure 19. *Upper left:* No backflow after 3 cc. injected by syringe. *Upper right:* Backflow, probably into excretory tubes (compare with Figure 8). *Lower left and right:* Further development of the backflow (17 and 21 cc.).

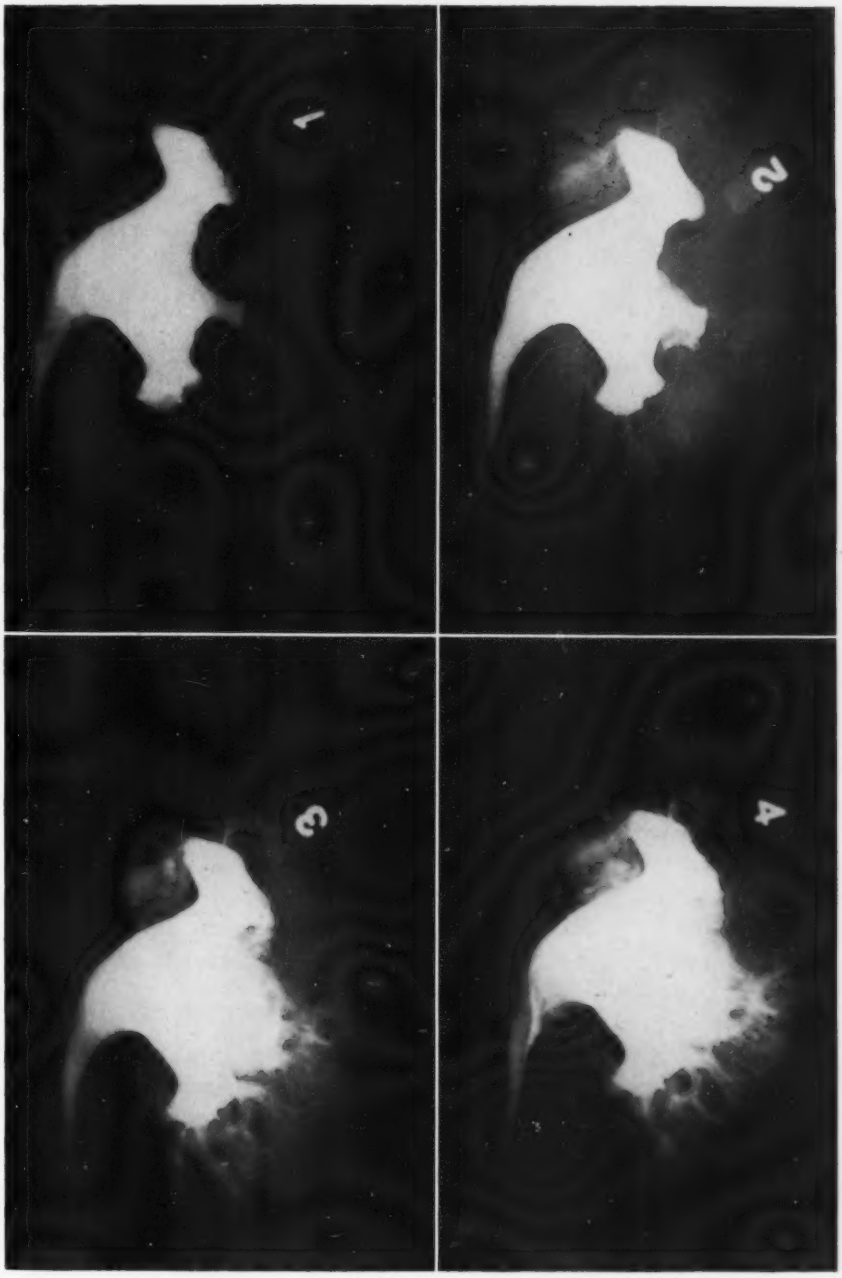


Fig. 21. Experiment VIII. Ureteral injection of neo-iopax by syringe. *Upper left:* Appearance after 6 cc.—no backflow. Pyelosinal and pyelotubular backflow are shown in the remaining films. Note also the fan-shaped extravasates such as already illustrated in clinical material as in Figures 8, 9, 10, etc. *Lower left and right:* Backflow after injection of 17 and 18 cc. of medium respectively. Addition of India ink to the opaque medium and subsequent histologic study of blocks of kidney showing the fan-shaped extravasates are suggested to provide evidence of the location of the fan-shaped extravasates.

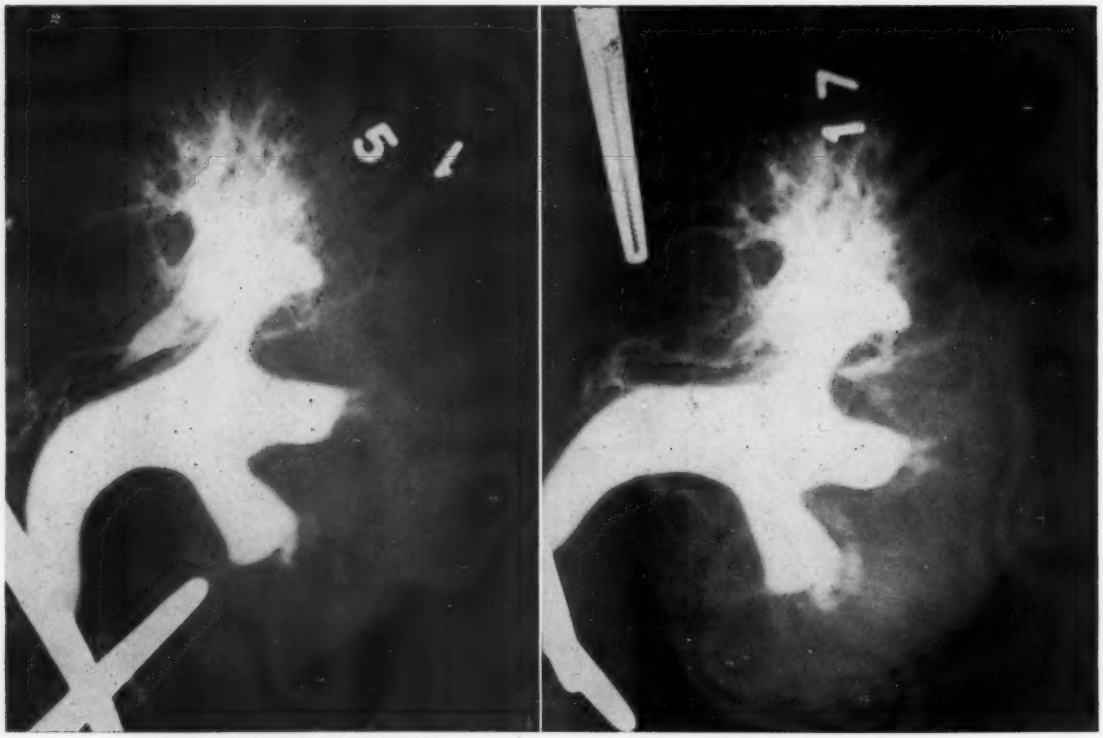


FIG. 22. Experiment IX. Ureteral injection of neo-iopax by syringe. *Left:* After injection of 15 cc. of opaque medium. *Right:* After injection of 17 cc. of opaque medium. Pyelosinal, pyelointerstitial, pyelovenous, and pyelolymphatic backflow are illustrated. Compare with the fan-shaped extravasates of Figures 1, 2, 4, 6, 8, 9, 10, 11, 21, and 25. In those illustrations, the backflow extends fully to the capsule whereas here there is a clear zone between the capsule and the periphery of the extravasate.

and veins. Actually, this was done on only one kidney of a pair (Fig. 27) and the other kidney was saved as control. The illustrations show that pyelovenous backflow occurred bilaterally but occurred much earlier in the kidney which was used as control (Fig. 28).

In all of these injection experiments, care was taken to avoid sudden increases in pressure. It is for this reason that so many films were taken. With each slight increase in the amount of contrast medium injected, another exposure was made. Thus, the experiments here simulated excretory urography in that there was a constantly but slowly increasing pressure in the "closed system." In some experiments, exposures were made whenever ½ cc. of additional contrast medium was injected. In other cases the exposures were made each time 1, 2, or 3 additional cc. of the medium was injected.

Both oil and water soluble media were utilized including skiodan, neo-iopax, pantopaque, diodrast, and urokon.

TECHNIQUE OF EXPERIMENTS

Fresh specimens were obtained and these were utilized immediately in most cases. The kidneys were removed with as little damage as possible to pelves, ureters, blood vessels, and perirenal fat. An attempt was made to preserve the lymphatic vessels running to the aortic lymph nodes. Plain or bulb tip catheters were inserted into the lumen of the structure to be injected, and held in place with elastic bands which prevented return flow of the injected contrast medium around the catheter tip. In early experiments, gravity flow was employed to keep the injection pressure at a minimum. This was found to be unnecessary. Injection was done under fluoroscopic guidance

and multiple spot films were made. For such a method, gravity flow proved to be undesirably tedious. Furthermore, all changes which developed under the better controlled injection by syringe could be recorded instantly with the multiple spot film method. The subject-film distance was minimized by holding the cassettes directly against the kidney specimens.

The resulting films demonstrated pyelovenous (Fig. 19, 20, 22 and 25), pyelotubular (Fig. 12, 20 and 21), pyelosinal (Fig. 12, 13, 14, 17, 19, 21, 22, 26 and 27), pyelolymphatic (Fig. 13, 17, 19 and 22), pyelointerstitial (Fig. 19 and 22), and pyeloarterial (Fig. 21 and 28) backflow. The exact technique utilized by previous authors has not been found in their published works. I believe fluoroscopic control and care to leave abundant perirenal fat are significant points by which my technique differs from previous authors.

DISCUSSION

The arterial pattern shown by opaque medium injected into the renal artery is illustrated in Figures 15, 18 and 24. An entirely similar pattern is revealed in Figure 21 of Experiment VIII where the opaque substance was injected into the ureter only. The fan-shaped backflow pattern in Figure 21 corresponds well to the fan-shaped extravasates of Figures 1, 2, 4, 6, 8, 9, 10 and 11 from clinical cases. I believe this represents pyeloarterial backflow.

That pyeloarterial backflow has not been conclusively established from the work here presented is readily admitted. A further study which I was unable to carry out should still be undertaken and might contribute the necessary proof. The addition of India ink or similar substance to the opaque medium utilized for injection of cadaver specimens is suggested. Then, when a fan-shaped extravasate develops, extending from calyx to kidney capsule, blocks should be cut and a histologic check made as to the localization of the extravasate.

Olsson sought to disprove the occurrence of pyelovenous backflow in vital human kidneys by adding fluorescein sodium to the contrast medium in retrograde pyelography. In selected individuals, without renal infection, an attempt was made to induce backflow by gentle increase in pressure. Sometimes the renal pelvis was held full of the fluorescein contrast substance up to 20 minutes. The patient's head was held under a cover to which a fluorescent illuminator was fitted so that if any fluorescein from the kidney were to escape into the blood, evidence of this would be obtained by an observer stationed at the patient's head during the procedure. No fluorescence was observed in a study of 10 such patients. I recommend that this work be repeated. With proper experimental conditions, I an-

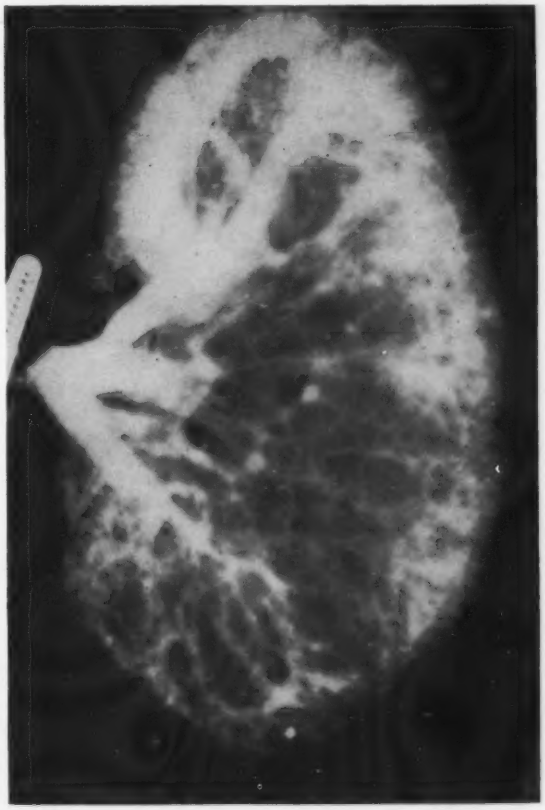


FIG. 23. Experiment X. Intravenous injection of neoiopax into the renal vein shows some perirenal vessel filling. There is no evidence of lymphatic filling even at the hilus. Lack of sharpness is due to beginning spread of the contrast medium from escape through vessel walls into the kidney substance. This is a reversal of pyelovenous backflow.

ticipate fluorescence will be seen in the conjunctivae, lips, and mucous membranes of patients with backflow such as shown in Figures 1, 2, 4, 6, and 8–11.

Deaths and less serious reactions which occur in excretory urography are a constant source of worry to the radiologist. Several times I have observed reactions in which the patient manifested signs of shock but no skin wheals, itching or other signs of sensitivity. Liedholm[1] observed backflow and accompanying decrease in blood pressure in animals, sufficient to cause shock. Fall in blood pressure was due to the presence in the urine of substances with hypotensive activity. If there be any relation between Liedholm's experiments and the reactions I have observed and mentioned above, a correlation can perhaps be proved by proper roentgen studies.

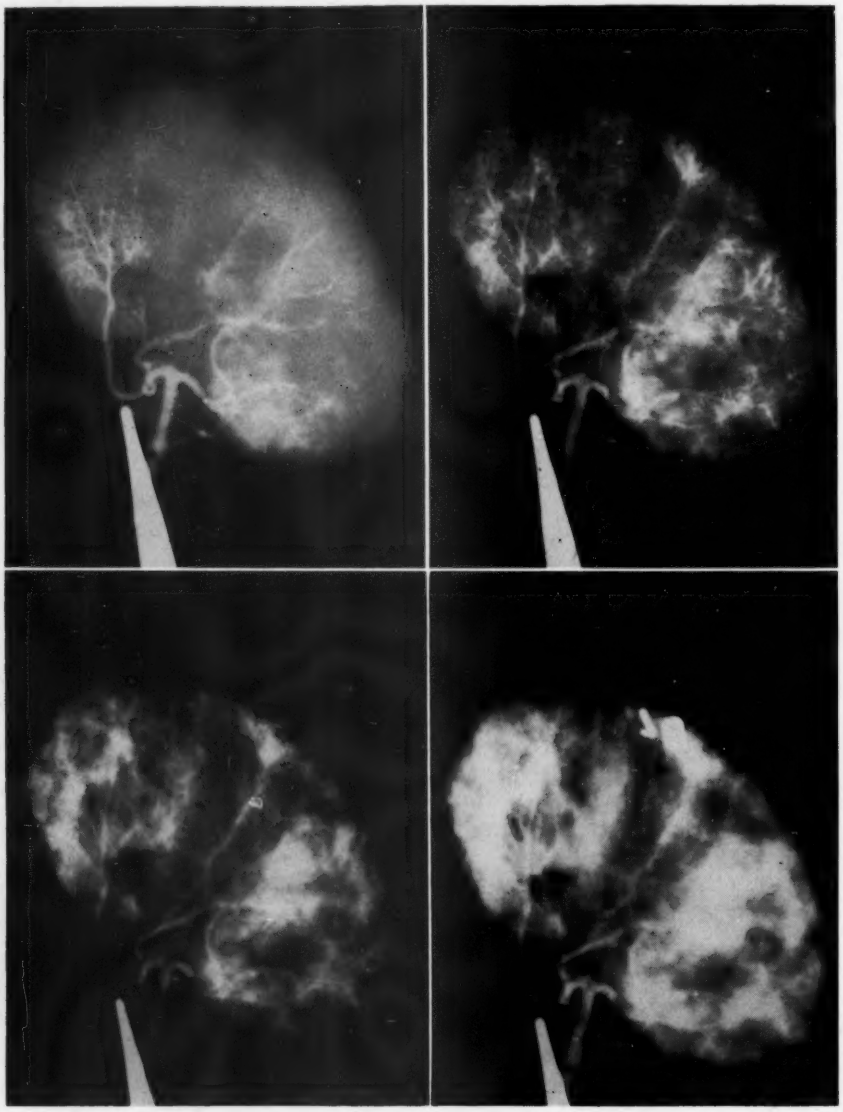
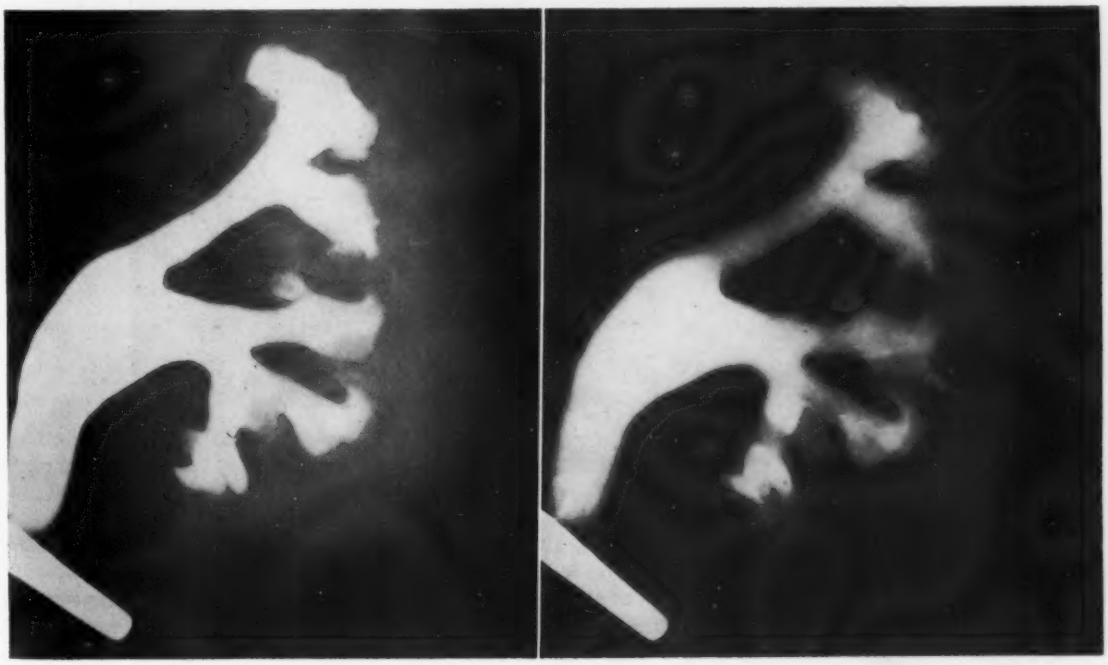


Fig. 24. Experiment x. Arterial injection with neo-iopax shows fan-shaped cortical patterns which recall the extravasates seen in Figures 1, 2, 4, 6, 8, 9, 10, 11, 21, and 25. The following amounts were injected: upper left—1 cc.; upper right—7 cc.; lower left—14 cc.; and lower right—18 cc. This experiment suggests that the observed fan-shaped extravasates may represent pyeloarterial backflow.

Fig. 25. Experiment xi. Ureteral injection of urokon by syringe. This kidney had been refrigerated for 8 hours before study. Pyelovenous backflow started with the first cubic centimeter injected. Later pyelosinal and other backflow forms began. Pyeloarterial backflow is suggested in the lower pole. Note filling of some perirenal fat vessels.

Many illustrations in the literature which purport to show pyelovenous backflow in life are questionable. The theoretic problem is to obtain sufficient concentration of opaque medium in the vein, despite constant dilution by blood. The newer media of greater per unit density should make the demonstration easier. Certainly, if pyeloarterial backflow can be recorded, so can pyelovenous backflow. Therefore, I

Fig. 26. Experiment xi. Ureteral injection of urokon by syringe. This is the opposite kidney to that shown in Figure 25. This kidney was out of refrigeration 45 minutes before injection whereas the opposite one was injected promptly after removal from refrigeration. The differences in backflow are probably not from this difference in handling. Note that backflow was later in development, not commencing until 7 cc. of medium had been injected (left). Even at 10 cc. (right) backflow is early. The temperature of the kidney is probably less important than the amount of water (urine?) retained in the pelvis at the start of injection, helping to distend the calyces and to add to the intrapelvic pressure.



suggest that special emphasis be given to finding pyelovenous backflow in patients who show evidence of a drop in blood pressure but no other signs of sensitivity during excretory urography. Cone-down films of the kidneys should be made at once and at intervals until external compression over the ureters is released.

CLINICAL APPLICATIONS

The risk of hematogenous spread of infections is a theoretic possibility with pyelo

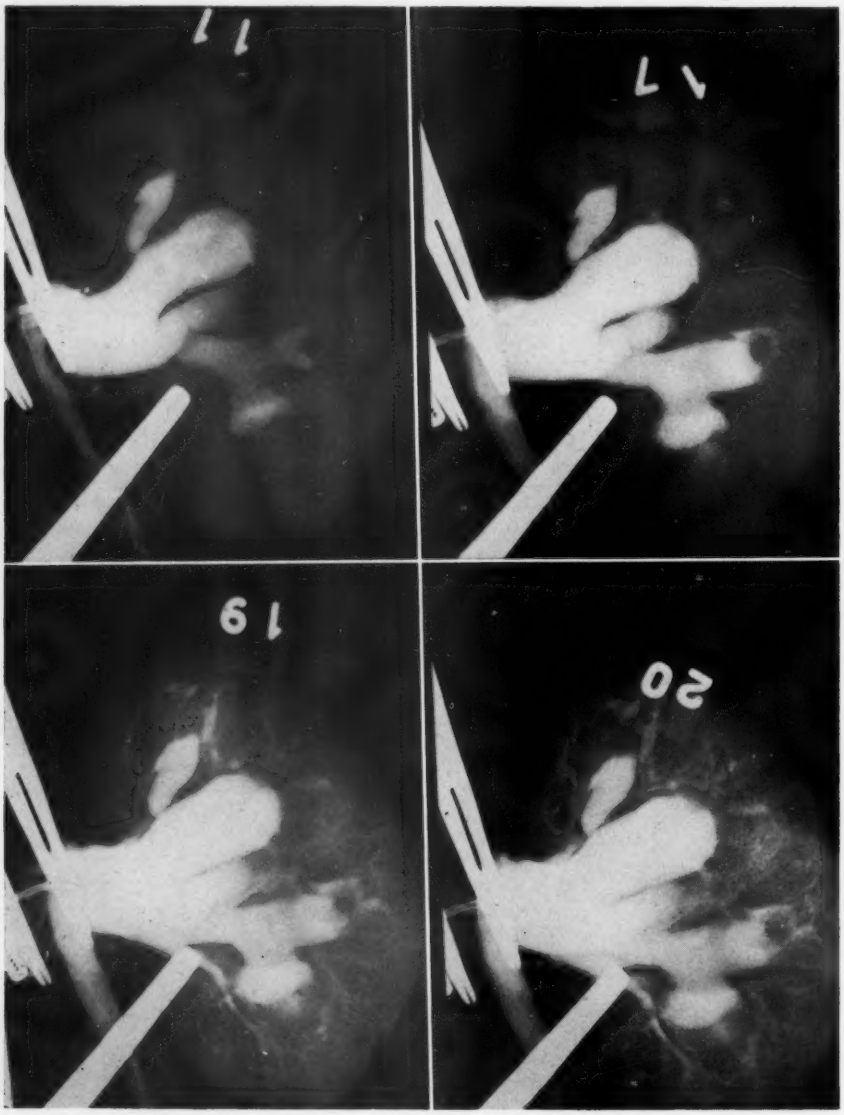


Fig. 27. Experiment xii. This kidney and the one in Figure 28 are from the same individual; they were on ice for four hours before injection. As in Experiment xi, one kidney was injected 45 to 60 minutes before the other and, thus, was colder. The following were the findings, depending on the amounts of diodrast injected at the time the respective exposures were made: upper left (11 cc.), beginning of pyelosinal backflow at the tip of the next to lowest calyx; upper right (17 cc.), further development of this together with backflow in the lowermost calyx and elsewhere; lower left (19 cc.) and lower right (20 cc.), further development of various forms of backflow. The kidney in this figure had both the renal artery and the renal vein injected with citrated blood prior to the start of the ureteral injection of diodrast. Before each increase of the amount of the contrast medium, the pressure on the venous side was increased by injection of an additional amount of blood.

venous backflow. This possibility makes worthwhile any project which may result in demonstrating whether pyelovenous backflow does occur in excretory urography.

Pyelolymphatic backflow is well established and frequently seen. The risk of lymphogenous spread of infection and neoplasm is a theoretic possibility in pyelolymphatic backflow.

If pyeloarterial backflow does occur in retrograde pyelography, as suggested here, then the risk of hematogenous spread of infections by pyeloarterial backflow is an added risk in retrograde pyelography.

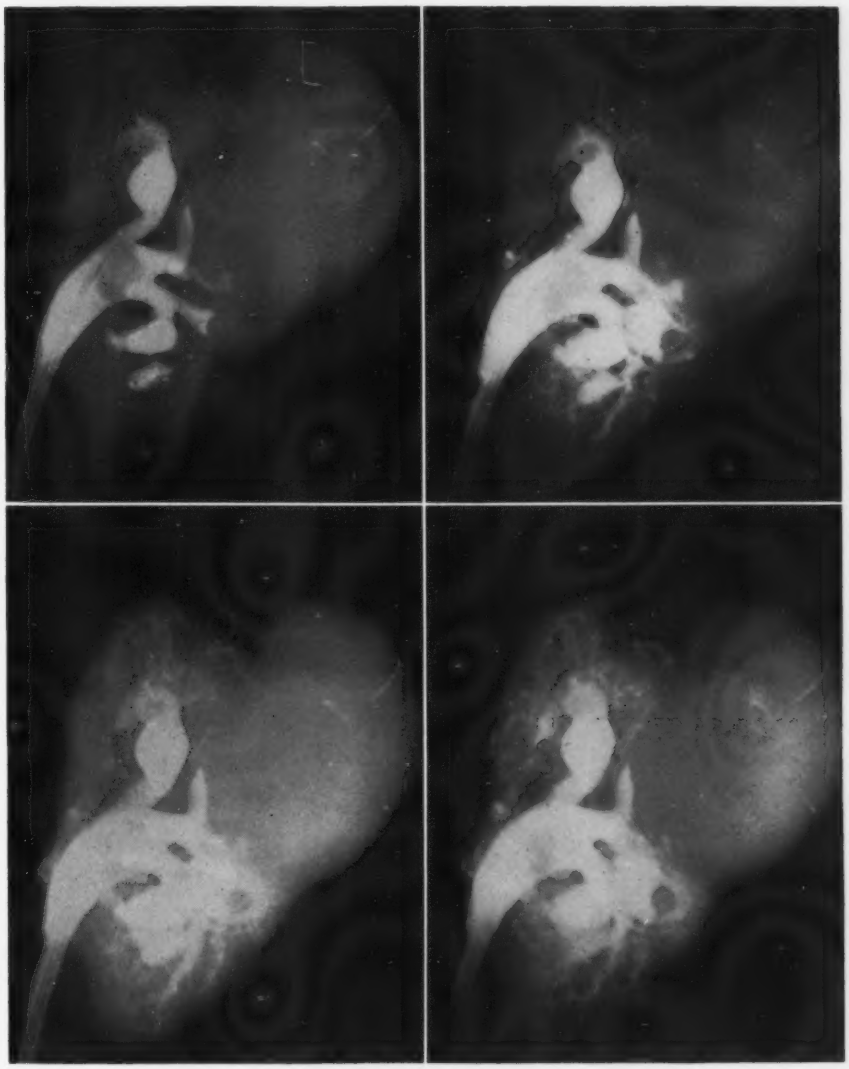


Fig. 28. Experiment xii. This is the mate to the kidney in Figure 27. This kidney was warmer than the opposite kidney at the time of injection. This contrasts with the situation of Experiment xi where the colder kidney showed backflow earlier. Here in Experiment xii the warmer kidney shows the backflow earlier, but conditions have been changed in another respect. In the kidney of Figure 27, the arteries and veins had been injected with blood. In the kidney of this figure, no arterial or venous injection was made and the ureter was injected in the manner similar to that of the kidney of Figure 27. Note the advanced stage of the backflow for 6 cc. (upper right). Clearly this experiment would have to be repeated several times before a definite conclusion could be drawn but there is the possibility that the earlier backflow is due to lack of resistance from low pressure in the arteries and veins. The type of fan-shaped, diffuse cortical filling seen on some of the clinical films is again shown on the 9 cc. film here (lower right).

The possible spread of tumor or infection within the kidney by any form of backflow is readily evident. Cadaver experiments (Fig. 18 and 23) emphasize the ease with which opaque material may pass into the perirenal vessels. Pyelosinal backflow is seen to escape from the hilus (Fig. 13 and 17). These observations point up the possibility of spread of intrarenal disease beyond the capsule as a result of backflow. Under certain circumstances, then, a simple diagnostic procedure may be transformed into a measure for spread of disease within the body.

SUMMARY

Pyelorenal backflow has clinical importance because the extravasation of urine from the pelvis into the kidney substance or into arteries, veins, or lymphatics may be attended by the spread of disease. Dissemination of neoplasm or infection is a possible concomitant of backflow. In as much as backflow is more frequent in retrograde than in excretory urography, retrograde studies are more to be avoided where neoplasm or infection are suspected. The comparisons of clinical roentgenograms demonstrating backflow with experimental material suggest that even pyeloarterial backflow may occur.

CONCLUSIONS

1. Pyelorenal backflow of all clinical types can be demonstrated in postmortem kidneys.

2. The frequency of pyelolymphatic backflow is related directly to the pressure of injection in retrograde studies and the effectiveness of external ureteral compression in excretory studies.

3. Pyeloarterial backflow may be produced experimentally in postmortem kidneys and clinically in retrograde studies.

4. All backflow is potentially harmful because it may serve to disseminate disease within and beyond the kidney.

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OPACIFICATION OF THE RENAL PYRAMIDS IN INTRAVENOUS UROGRAPHY

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THE phenomenon of extracalyceal opacification in retrograde pyelography is commonly observed and is well understood. Various types of pyelorenal backflow and the roentgenologic characteristics of each have been described by Narath[4] and Braasch and Emmett.[2] This report deals with a phenomenon of a somewhat similar appearance which has been observed in intravenous urography.

There has been a strong recent trend toward the use of intravenous substances which afford very dense opacification of the urine in the collecting system of the upper urinary tract. This has not only provided excellent visualization of the pelves and calyces, but has furnished the requisites for the opacification of structures proximal to the calyces. It is our purpose to illustrate and discuss the patterns sometimes produced when the urographic medium opacifies the tubules and ducts in the renal medulla. Unless these shadows are understood they may lead to misinterpretation.

ANATOMY

Human kidneys contain from four to eighteen medullary pyramids, the apices of which project into the minor calyces. The pyramids are usually arranged either anteriorly or posteriorly to the coronal plane, so that when a kidney is sectioned longitudinally the pyramids are cut obliquely and the gross anatomy is not well shown. A diagrammatic representation of renal anatomy is shown in Figure 1. The occurrence of numerous pyramids explains the calyceal complexity found in some kidneys. Each pyramid is made up of collecting tubules which coalesce into larger papillary ducts and enter the minor calyx at the apex (known as the renal papilla). Although the number of papillary ducts per papilla ranges from seven to fifty,[2] an average papilla contains from ten to twenty-four. The ducts are generally straight, about 20–22 mm. in length,[1] and extend outward in a fan-like manner.

Ordinarily the collecting system proximal to the minor and major calyces is not seen on intravenous urographic study. It appears, however, that under certain circumstances and in some individuals the collecting tubules and papillary ducts may become opaque in intravenous urography. This has been reported as extravasation in a patient studied by Castano[3] and is mentioned without comment by Narath.[4]

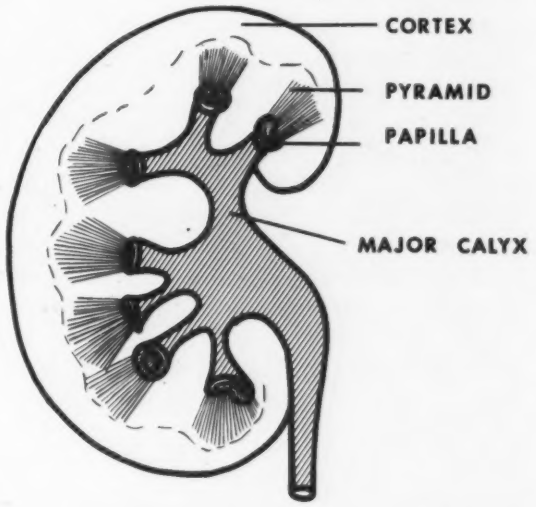

FIG. 1. Diagrammatic presentation of the gross anatomy of the kidney. The renal pyramids project into the calyces. The papillary ducts empty into the calyces at the papillae.

ROENTGENOLOGIC APPEARANCE

Usually the appearance of the pericalyceal opacification is similar to that of

* Deceased.

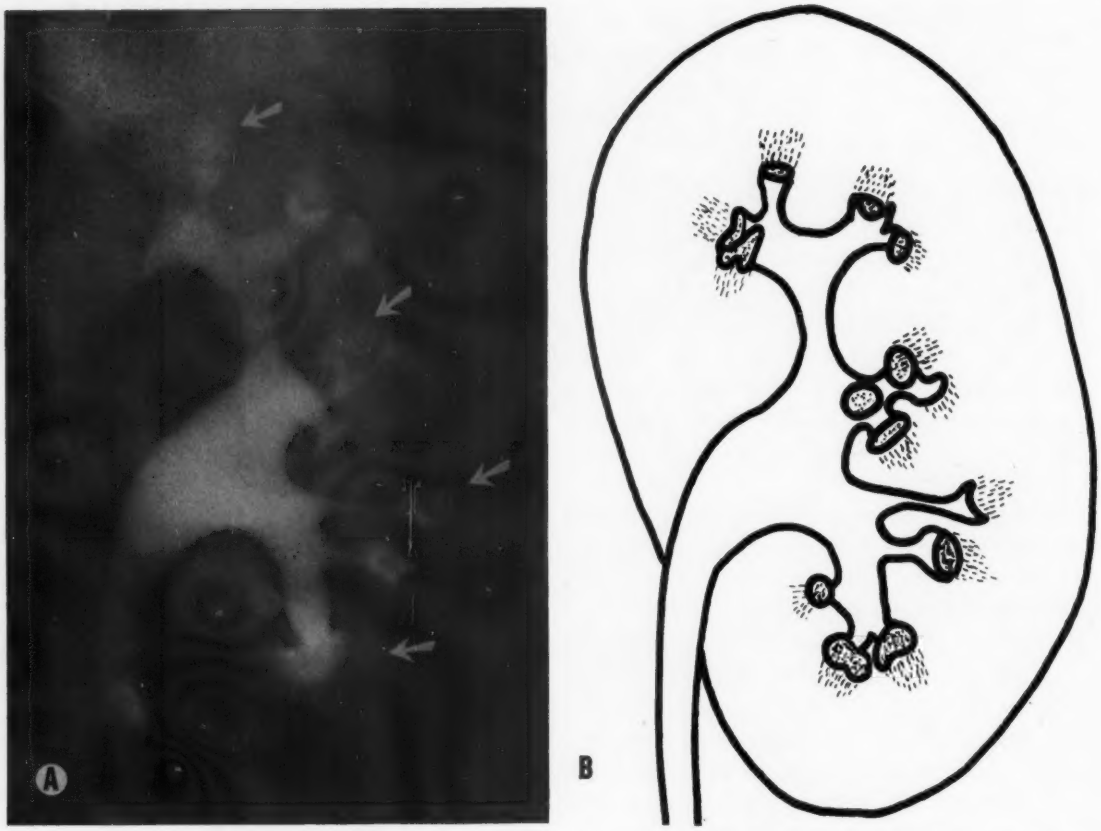

FIG. 2. Opacification of renal pyramids. (*A*) Intravenous urogram. (*B*) Line drawing of the same kidney.

the better known "pyelotubular reflux" commonly seen in retrograde pyelographic studies. The most frequent appearance is a brush-like or flame-shaped flare of opacity extending outward from the minor calyces. The typical pattern is illustrated in Figure 2. The shape of the parenchymal shadows is variable since some calyces are seen in profile, others are seen end on, and in complex systems two or more may be superimposed. The size and extent of the pyramidal opacity are also quite variable. When there is only slight opacification of the renal pyramids, the calyces may appear merely to be fuzzy and indistinct. Figures 3 and 4 illustrate this type of opacification. When there is more extensive opacification of the pyramids, the appearance may become complex. In Figure 5, for instance, the shadows resemble clusters of bells or flowers. Occasionally, the phenomenon may simulate clubbing or ulceration of calyces. In contrast to this the sharply rounded opaque collections in Figure 6*A* simulate calycine diverticula. In one young woman (Fig. 7) the opacification was so atypical that renal tuberculosis was seriously considered.

In most cases the phenomenon is bilateral and involves multiple pyramids, giving rise to no difficulties in diagnosis. We have observed 20 cases showing the finding bilaterally in the last year. In 3 instances it was unilateral, and twice it appeared to involve only one lower-pole calyx. We have observed the phenomenon in patients of all ages and both sexes.

DIFFERENTIAL DIAGNOSIS

The principal identifying characteristics of pyramid opacification may be summarized as follows:

1. The shape and appearance are usually typical (see text).
2. Renal concentration is excellent.
3. The phenomenon usually appears on

Fig. 4. Example of pericalyceal "sunburst" appearance on the 15-minute film. This was absent at 45 minutes. Such a finding might be mistaken for clubbing of calyces.

the 10- and 20-minute films when the ureters are compressed. It decreases or disappears when compression is removed.

4. It has not been found later than 45 minutes after injection.
5. It is usually bilateral and involves multiple pyramids.
6. Review of previous urograms usually reveals the same finding (*i.e.*, it tends to occur repeatedly in certain individuals).

Fig. 3. Opacification of pyramids produces haziness of calyces suggestive of loss of outline (hypaque urogram).

Fig. 5. Uropaque in pyramids presenting "flower-like" appearance. Subsequent films without ureteral compression showed normal calyces.

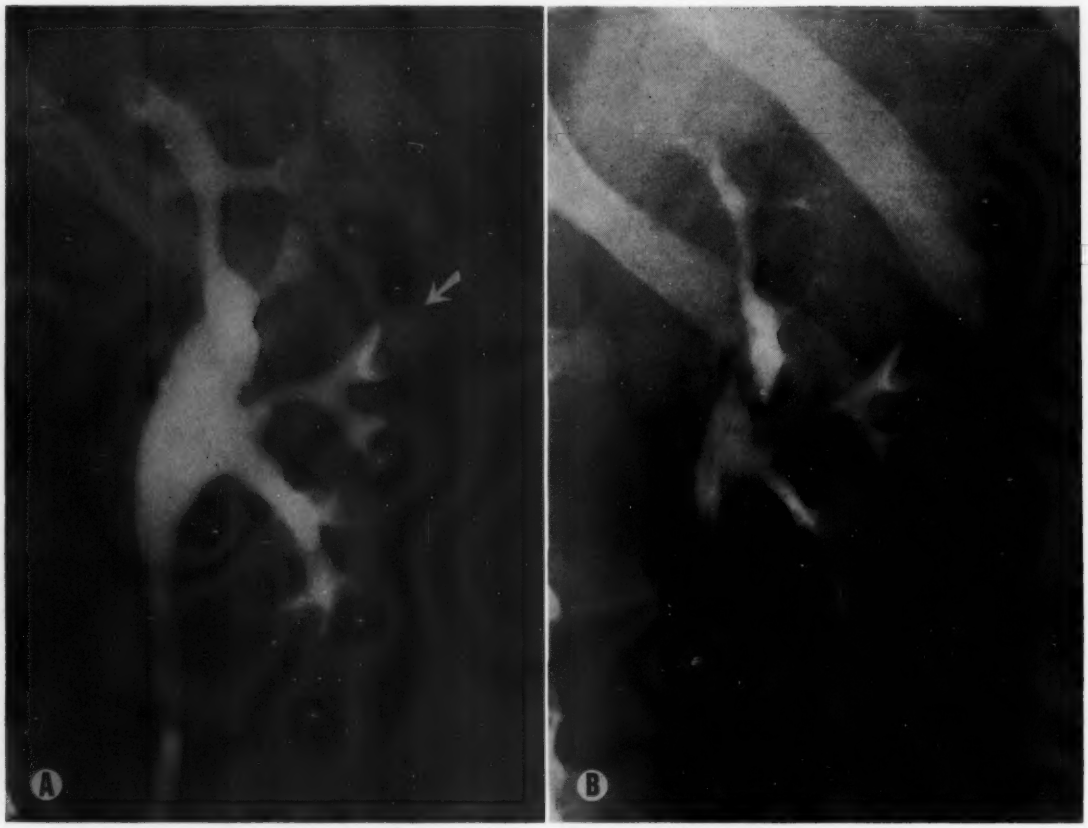

Fig. 6. Intravenous hypaque urogram. Changing pattern of pyramid opacification. (*A*) Ten-minute film with compression. There is a marked resemblance to calycine diverticula. (*B*) Forty-five-minute film without compression.

The brush-like shadows described above should offer no serious diagnostic problems if the possibility of their occurrence is kept in mind. Under some circumstances, however, it may be necessary to differentiate them from the following: (1) calyceal diverticula; (2) caliectasis; (3) calyceal ulceration; (4) renal calcinosis or calculi; and (5) tuberculosis or papillary abscess.

The plain film is of great value in excluding the presence of opacities such as renal calculi or parenchymal calcinosis. Films after the removal of ureteral compression are, of course, essential. Delayed films made 45–60 minutes after injection show disappearance of the phenomenon, thus permitting differentiation from permanent or anatomic abnormalities. It is wise to have films marked in such a way that those made with ureteral compression are identifiable.

Factors Contributing to Opacification of the Pyramids

In the first 8 patients showing this phenomenon, hypaque was used together with external ureteral compression. It was our original impression that we were dealing with a form of "pyelorenal reflux." This simple explanation of the phenomenon, however, failed to suffice when the opacification was observed in patients without ureteral compression or obstruction and when it was seen unilaterally in patients with bilateral compression.

The phenomenon is most often seen when very dense substances such as hypaque, renografin, or miokon are used, but it has also been identified on review of films made using neo-iopax. It is seen most frequently in thin young adults with normal concen-

Fig. 7. Ten-minute film, intravenous urogram. The granular pattern of the lower-pole calyces suggested the possibility of tuberculosis.

trating power who have films of excellent technical quality.

It appears that the factors which contribute to the production of the phenomenon on roentgenograms are: (a) good renal concentrating power, (b) high iodine content in the tubular filtrate, (c) relatively high intracalyceal pressure, and (d) films of excellent technical quality. In addition to these, there may be an anatomic factor (such as dilated collecting tubules) which characterizes the kidneys of patients showing this phenomenon.

DISCUSSION

There is little doubt that modern urographic opaque media permit visualization of antomic structures which could not previously be seen. It appears that, when there is good concentration by some kidneys, there is sufficient iodine in the larger renal tubules and in the papillary ducts to render them opaque. Probably, effective ureteral compression elevates the intracalyceal pressure enough to favor the retention of the opaque material within the ducts. Since the phenomenon of opacification of the pyramids is not wholly dependent upon ureteral compression, however, it cannot be attributed to high intracalyceal pressure or pyelorenal reflux alone.

It is curious that the phenomenon is not found in practically all thin patients with good renal function when very dense uropaques are combined with effective ureteral compression. The fact that it is not more frequently seen suggests that the ducts of some patients are larger or more readily opacified than the ducts of others. This concept is strengthened by the finding that when multiple urograms were done on some of the patients in this series, opacification of the pyramids occurred on each examination.

The clinical significance (if any) of opacification of the renal pyramids in urography is not known. Review of the 25 patients forming the basis of this report discloses no consistent abnormalities in urinary findings, blood chemistry, cystoscopic findings, blood pressure, or symptoms. Most of the patients had no history of previous urinary tract abnormalities. Final urologic diagnoses were established in our series of patients as follows: granular trigonitis (2 patients), cystocele (1 patient), small ureteral calculus (1 patient), prostatic enlargement (1 patient), bladder-neck obstruction (1 patient), unexplained hematuria (1 patient), no urologic disease (18 patients).

Whether or not the phenomenon is related to previous urinary tract disease is conjectural. Pathologists sometimes find dilated tubules and papillary ducts in kidneys which have been involved by certain infectious or obstructive diseases.[1] It is possible that the kidneys of the patients in this report are in some manner abnormal or unusual, and that for this reason the parenchyma is prone to opacification on intravenous urography.

SUMMARY

Opacification of renal pyramids occurs occasionally in intravenous urography. The uropaque collections may simulate renal abnormalities. The phenomenon is intensified by the use of iodine-rich uropaques combined with ureteral compression. It is possible that such opacification indicates dilatation of the papillary ducts and renal tubules. Recognition of this type of pyramidal density should permit its differentiation from organic disease involving the renal calyces.

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URACHAL CYST WITH CALCULI

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DALLAS, TEXAS

ANOMALIES of the urachus are among the rare anomalies which may be, or may become, clinically significant. Sterling and Goldsmith[10,11] reported only 2 cases in over 300,000 general hospital admissions. The present case is the only one on record in over 55,000 admissions to the Veterans Administration Hospital, Dallas, Texas. The incidence in adults is very low in comparison to the incidence in children.

The urachus persists usually as a fibrous cord, the middle umbilical ligament, which may have a potential lumen lined with urothelium or squamous epithelium. Variations which may exist are: (1) The tract may be open, communicating with both the urinary bladder at its dome and the umbilicus, in which case varying amounts of urine will be discharged at the umbilicus. These cases are usually apparent at birth, or soon thereafter, and the diagnosis is made very early in life. (2) The tract may be open only at the umbilicus with the formation of a urachal sinus. (3) The tract may be open only at the bladder end and may appear as a diverticulum of the fundus of the bladder. (4) The tract may be closed at both ends and may have an open lumen in one or more places in between with the formation of urachal cysts. Through increase in size and with increase in intracystic pressure, the type 4 variations may be converted into one of the other types or may rupture through the peritoneum or externally.

Most lesions of the urachus become apparent during early childhood and usually by the age of five. Sterling and Goldsmith [10,11] in describing their 2 cases stated that only 26 proved cases of urachal tract involvement have been reported in which the signs and symptoms appeared after the age of five.

When lesions of the urachus become symptomatic in the adult, it is due to complications in the urachal structures, often associated with complications in the urinary system, especially subvesical obstruction and urinary tract infection. Complications of urachal fistulae and cysts include (1) benign and malignant tumors, (2) abscesses, (3) tuberculosis, and (4) calculi. Carcinoma has been the most frequent complication and stones the rarest complication mentioned in the literature.

Six cases of urachal concretions have been recorded since 1877. Dreyfuss and Fliess[3] recorded the case of an eleven year old white girl with a fetal bladder containing a uric acid stone 4.0 by 1.8 cm. This child died from overwhelming toxemia 36 hours after her hospital admission. Wyatt and Lanman[13] reported calcification and ossification of the remnant of a urachal cyst at the bladder wall in a boy of seven. This is the only case found in a review of the literature in which it is clear that a urachal calculus was seen roentgenographically. Siddall[9] cited the presence of a steatolith in an extension of a urachal cyst in a twenty-four year old Chinese girl. Ward[12] found an hourglass type of calculus in the urachus of a Hindu. Weiser (1906) reported a 70 grain calculus removed from a suppurating urachal cyst. There is mention of 14 cases of urachal concretions in the literature prior to the twentieth century. Phillips, in an article in Todd's "Cyclopedia of Anatomy and Physiology" (1835), relates the case of Boyer who in 1787 exhibited a bladder with a distinct urachal canal containing calculi. This is the only reference found in the literature of multiple urachal stones.

REPORT OF A CASE

The case being reported is that of a sixty-one year old colored male with five stones measuring 1.4 cm. in greatest diameter contained within a urachal cyst measuring 11 cm. in greatest diameter. The cyst communicated with the bladder

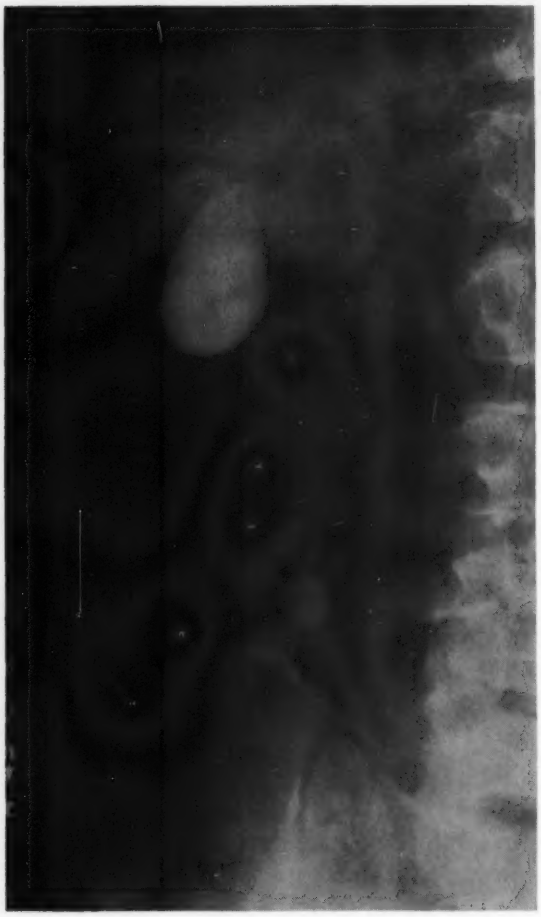

Fig. 1. Posteroanterior roentgenogram of right half of the abdomen with telepaque outlining the gallbladder. Four of the 5 urachal calculi are visible above the crest of the ilium medially. The shadow over the ilium laterally is barium in a diverticulum of the cecum and this became a useful landmark in other examinations.

through a small tract opening into the fundus.

The patient entered the hospital on December 30, 1955, for treatment of bilateral chronic glaucoma. Systemic review revealed symptoms of straining to start the urinary flow for about six months prior to admission. The patient had noticed a vague mass in his lower abdomen for several months, but this information was not obtained until after the stones were discovered roentgenographically. He denied ever having any degree of tenderness in the lower abdomen and denied any symptoms of urinary tract infection. On physical examination, no abdominal masses were palpable and none became palpable during his hospitalization. Admission laboratory studies revealed: white blood cell count 10,050, with a differential of 76 per cent neutrophils and 24 per cent lymphocytes; red blood cell count 4.27 million; and hemoglobin 11.8 gm. per cent. Urinalysis revealed: color, amber-hazy; alkaline reaction; specific gravity 1.012; albumin, one plus; sugar, negative; microscopic, innumerable red blood cells and white blood cells. Serologic tests for syphilis were negative.

Fig. 2. Right lateral decubitus roentgenogram of abdomen showing the calculi partially superimposed on each other far to the right. The lateral shadow again is barium in a cecal diverticulum.

During the evening of January 2, 1956, the patient developed abdominal pain originating just below the umbilicus, radiating to the epigastrium. He developed nausea, vomiting and diarrhea and become greatly distended. The abdomen was tympanitic above the umbilicus and dull from the umbilicus to the pubis as though the bladder were greatly distended; however, catheterization yielded only 200 cc.

Fig. 3. Left lateral decubitus roentgenogram of abdomen showing the calculi far to the left. Note the distance they have moved from the cecal diverticulum.

of urine. Flat and upright anteroposterior roentgenograms of the abdomen, supplemented by a lateral roentgenogram with a horizontal beam, on January 3, 1956, showed numerous loops of small and large bowel distended with fluid and gas. On this examination, 5 radiopaque stones were seen grouped together just to the right of the midline in the lower abdomen. They always occupied a dependent position and on the upright roentgenogram they dropped just below the brim of the pelvis. Repeat urinalysis on January 3 showed gross white blood cells. The patient was started on chemotherapy and antibiotics. A Levine tube was placed in the stomach and Wangensteen suction was started. The patient's gastrointestinal symptoms quickly subsided, and subsequently he had no subjective complaints. His urine gradually cleared.

Interest then centered on establishing the identity of the right lower quadrant lithiasis. Right and left lateral decubitus roentgenograms with a horizontal beam showed that the stones would move from a position near one anterior iliac spine to a similar position on the opposite side. Roentgenograms taken with a horizontal beam and with the patient prone showed the stones far anteriorly; with him supine, they were far posteriorly. Cholecystography showed no significant abnormality. Barium enema and small bowel studies outlined a cystic mass in the right lower quadrant and practically excluded the possibility of stones in a Meckel's diverticulum. Excretory urography showed normally functioning kidneys occupying their usual position. The ureters were not dilated or displaced. None of the contrast medium on any of the examinations entered the cystic mass. It was noted that the outline of the mass seemed to be confluent with the outline of the bladder, making a dumbbell configuration. The various roentgen examinations clearly demonstrated a 12 cm., movable, cystic mass just to the right of the midline in the lower abdomen, containing 5 freely movable radiopaque stones. During the study of this patient, the various possibilities

Fig. 5. Anteroposterior roentgenogram of the abdomen during a small intestine series. The cyst is well outlined by the small intestine and ascending colon. The dumbbell configuration formed with the urinary bladder is apparent on this examination.

Fig. 4. Excretory urogram showing relationship of the right kidney to the cyst.

Fig. 6. Urachal cyst removed at surgery. The pedicle communicated with the fundus of the urinary bladder.

considered included stones in an ectopic kidney, gallstones, hydroureter, Meckel's diverticulum, diverticulum of the urinary bladder, urachal cyst, omental cyst, mesenteric cyst, and dermoid cyst. After the studies were completed, diverticulum of the urinary bladder and urachal cyst were the first two choices in the differential diagnosis. Most of the other possibilities had been ruled out. Unfortunately, cystograms, which had been suggested, were not obtained prior to operation.

At surgery on January 26, 1956, a urachal cyst was removed intact with its connections to the umbilicus and urinary bladder.

The pathological report by Dr. Martha S. Meredith was as follows: Gross: A large cyst measuring, in extended state, 11 by 8 by 8 cm. Attached to the cyst was a pedunculated-like stalk that was 2.5 cm. in length. The area of this stalk opposite to the cyst contained a segment of bladder mucosa that was 2 cm. in greatest diameter. The bladder mucosa was smooth, glistening and transparent and on palpation contained a small, 3 mm., firm, white nodule. Connecting from this segment of urinary bladder mucosa and the lumen of the large cyst was a sinus tract that measured 2.5 cm. in length and varied in circumference from 5 mm. to 20 mm. This sinus tract was covered with smooth, glistening mucosa that resembled the urinary bladder mucosa except for the small, firm nodule. The cyst was filled with approximately 300 cc. of a clear, straw-colored fluid and contained 5 oval, smooth stones of approximately equal size that measured 14 mm. in greatest diameter. These stones were of a grayish tan color. The cyst wall varied in thickness from less than 1 mm. to areas that were 5 mm. in thickness. The external surface of the cyst contained a number of engorged vessels and was otherwise covered with some fairly smooth but opaque serosa. The lumen of the cyst showed a number of fine trabeculae that radiated from a few central points. At these central points, the lumen was fairly smooth, but in scattered small areas, it was very finely granular. The segment of urinary bladder mucosa was surrounded by a layer of urinary bladder wall containing smooth muscle that measured approximately 7 mm. in width. These muscle fibers were also present, surrounding the described sinus tract leading into the cyst. In this area the smooth muscle fibers lay parallel to the lumen of the sinus tract and radiated at the periphery of this cyst for a short distance, becoming narrow in the peripheral areas and extending out a distance of 1.5 cm.

Microscopic Examination: The cyst was

Fig. 7. Urachal cyst opened and showing the contained calculi.

lined in part by squamous epithelium and in part by urothelium, with a gradual transition between each segment. The cyst wall contained some scattered lymphocytes in a dense fibrous tissue in which scattered bundles of smooth muscle were present. The sinus connecting the cyst with the bladder was lined by intact urothelium and a broad zone of smooth muscle bundles arranged in parallel rows with the sinus tract. Diagnosis: Urachal cyst.

Chemical analysis of one of the stones showed it to be composed of calcium carbonate and calcium phosphate.

The patient made an uneventful recovery from his surgery and is now awaiting transurethral resection for hyperplasia of the prostate.

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EXPERIMENTAL HEPATOSPLENOGRAPHY USING BARIUM SULFATE*

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HEPATOSPLENOGRAPHY has been retarded in its development and application largely because the substances used for this purpose are radioactive[7] or have been attended by serious toxic reactions.[1,2,3,4,11,13,16,18] These shortcomings have prompted us to search for a nonradioactive preparation which would make the liver and spleen radiopaque after intravenous administration and which would not produce undesirable reactions. The present report is a summary of our observations on the experimental use of suspensions of barium in rabbits, rats, and dogs.

It seemed reasonable to assume that after intravenous introduction the particles of a fine suspension of barium sulfate would be collected mainly in the reticuloendothelial tissues in a fashion similar to thorium dioxide sol (thorotrast). Although barium is considerably less radiopaque than thorium by virtue of its lower atomic number (56 as compared with 90), the former has the advantage of freedom from natural radioactivity. Moreover, barium sulfate is inexpensive, easily obtainable, and can be ground into relative fine particles for suspension. It was postulated that after a sufficient quantity of barium sulfate was deposited in the reticuloendothelial elements of the liver and spleen, radiopacity of these organs would follow. The results obtained confirmed these assumptions but still left much to be desired before the material could be used in man. We are encouraged to report our observations, nevertheless, because they demonstrate the potential usefulness of such metallic suspensions and may stimulate others to extend and improve this work.

EXPERIMENTAL MATERIAL AND METHODS

For the sake of brevity, our experimental data will be briefly summarized.

Preparation #1, consisting of colloidally ground USP barium sulfate suspended in distilled water, formed a crude suspension, with single particles up to 4 micra, but with aggregates up to 30 micra. This material produced immediate emboli to the lungs and could not be used.

Preparation #2, a 25 per cent suspension of smaller particles (the larger were allowed to settle) was tolerated without reaction by 4 rabbits. The material was given fairly rapidly intravenously, and liver and spleen opacification was obtained within 2 hours after injection. Two of these rabbits were kept under observation for over one year and remained healthy. These were sacrificed, and histologic studies revealed large deposits of barium sulfate (up to 50 micra) in the alveolar septa of the lungs, in the sinuses of the splenic pulp (in phagocytic cells), and in the phagocytes of the liver lobules. No other significant abnormalities were noted in these or in other organs.

The radiopacity of the liver and spleen did not diminish in either of these 2 rabbits over the entire period of observation.

Preparation #3, prepared by allowing 24 hour settling of larger particles, showed a maximum particle size of 3 micra, but frequent clumps up to 20 micra were found. However, this material could be given without reaction into the ear veins of rabbits.

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Fig. 1

Fig. 2

Fig. 1 and 2. This rabbit received 3.5 gm. barium sulfate per pound. Note intense opacity of liver and spleen. Opacification of the bone marrow of the hind limbs can be seen clearly on original films. The opacities in the spleen in Figure 2 are in the nature of rather discrete granules.

It was found that a dose of 0.5 gm. of barium sulfate per pound was necessary for satisfactory hepatosplenography. Doses (divided into daily injections) up to 3.5 gm. per pound were given and tolerated. Opacification was intense, with deposition in the bone marrow noted on the roentgenograms (Fig. 1 and 2).

This same preparation was given intravenously to 12 Wister rats, using various dosages. No reaction was noted, and here too good visualization of liver and spleen could be obtained with 0.5 gm. barium sulfate per pound. Strangely enough, a later series of the same material but from a different ampule proved extremely toxic to another group of rats. Their difficulty was obviously due to pulmonary emboli. Apparently this ampule contained aggregates of too large a size to be tolerated.

Preparation #4 was made with a suspending agent (methylcellulose), and with greater colloid grinding. This suspension was superior, with maximum particle size up to 3 micra, and only occasional clumps up to 10 micra. This preparation did not settle on standing. It proved quite safe and satisfactory in numerous rabbit experiments.

This latter preparation and other similarly made suspensions were employed in 14 experiments on dogs. It was found that these animals became ill during the injections, with weakness, nausea, and salivation and defecation. These symptoms all subsided within one half hour. It was found that these symptoms could be minimized, though not completely eliminated, by giving the material slowly and diluted and, also, by preliminary subcutaneous adminis-

tration of methantheline bromide (banthine) in doses of 0.5 mg. per pound of body weight.

Despite the reactions encountered, every dog recovered completely and rapidly and remained well.

Opacification of the liver and spleen in the dogs was disappointing. It took up to 1.0 gm. of barium sulfate per pound body weight to obtain even faint splenic opacification. Doses larger than this were not administered.

DISCUSSION

A theoretically ideal agent for hepatosplenography should fulfill the following criteria:

1. The particulate matter of the colloid or suspension should be of minimal toxicity, both in its original chemical state and in any metabolically altered chemical form.

2. The preparation should not cause intravascular occlusion by dint of particle size or agglutination of particles. This requires a suspension or emulsion of extremely fine particles.

3. The atomic number of the opaque agent should be high enough to permit opacification with minimal amounts of the agent.

4. The material should be free from radioactivity.

5. The deposited material should be eliminated from the body within a reasonable time.

6. The liver and spleen, and not the lungs, should receive the greatest percentage of the material.

7. The preparation should be a colloid or suspension of great stability; it should be able to be stored for long periods without physical or chemical alterations.

Thorotrast (colloidal thorium dioxide, 25 per cent) has been the only heavy metal preparation used successfully to date in both animals and humans to opacify the liver and spleen. Although employed extensively between 1930 and 1940, its current occasional use is limited to individuals with a short life expectancy because the combination of permanency of deposition and radioactivity has apparently been responsible for late appearing cancer and late developing hematopoietic suppression.[1,3,4,6,8,9,10,12,13,14,15,16,17,18]

The experimental data presented in this report indicate that barium, a nonradioactive metal with a lesser atomic number than thorium, is also capable of opacifying the liver and spleen when given in sufficient quantity. As with thorium, however, barium has the defect of more or less permanent fixation once deposited in the reticuloendothelial structures. This was demonstrated by the persistence of barium in the livers, spleens, and lungs of 2 rabbits for as long as one year. This long term deposition in these animals resulted in no obvious cellular damage. It is possible, however, that in other animals or in other species some damage would occur and might have eventually occurred in these animals had they been observed for a longer time. Moreover, the colloidal suspensions of barium sulfate which were prepared and used in these experiments were not entirely free of particle agglutination. This defect was considered basically responsible for the toxic reactions observed not only in the rats but also in the dogs to which these suspensions were given. This belief was supported by the findings in those animals whose tissues were examined after death as well as by the fact that injection of the suspending agents alone was without reaction.

Experimental studies by Gabrieli[5] showed that the distribution in the tissues of injected suspensions varies greatly with particle size. Using a radioactive chromium sulfate suspension with an average particle size of 1 micron, he found that 50 per cent of the radioactivity was in the liver, 3.5 per cent in the spleen, and 0.33 per cent in the lungs. Using suspensions of somewhat larger particles (2 to 4 micra), the liver counts were only 27 per cent, the spleen counts remained at about 3.5 per cent, while the lung counts rose to 11.5 per cent. Thus, raising the average particle size above 1 micron halved the concentration of

colloid in the liver and increased the lung concentration 35 fold. It is possible, therefore, that intravenous administration of a finer suspension of barium sulfate than we were able to prepare would not only prove less toxic but would also opacify the liver after even smaller total amounts than we used. Perhaps our failure to opacify the liver in dogs even after doses of 1 gm. of barium sulfate per pound body weight is also to be explained by failure to secure a suspension of sufficiently small particles.

It is to be hoped that a truly colloidal preparation of barium sulfate, or of a metal with a higher atomic weight, will eventually be produced. Such a preparation should be clinically satisfactory for hepatosplenography even though it will doubtless still have the shortcoming of permanency of deposition. It may be anticipated, moreover, that a suitable preparation of this type should prove of value in many other roentgenologic procedures where a chemically inert medium may be employed more safely than the presently used soluble iodide compounds. Among such procedures are arteriography, aortography, angiocardiography, and venography.

SUMMARY AND CONCLUSIONS

1. Barium sulfate given intravenously in the form of a suspension providing 0.5 gm. of barium sulfate per pound of body weight opacified the livers and the spleens of rats and rabbits within 2 hours after administration. The same material given in twice this amount produced only faint splenic opacification in dogs.

2. Particles of barium sulfate were observed to remain in the reticuloendothelial cells of the liver, spleen and lungs of rabbits for a year without evidence of significant cellular damage.

3. The barium sulfate suspensions used in these studies were not entirely free of particle agglutination and toxic reactions were frequent. Such reactions occurred most severely in dogs.

4. It is felt that particle size may be reduced and particle agglutination significantly corrected, or perhaps entirely eliminated, by improved methods of preparation. When this is achieved, suspensions of nonradioactive heavy metals should prove feasible for use in man to opacify the liver and spleen and for other roentgenologic functions.

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PRIMARY RETROPERITONEAL TUMORS*

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PRIMARY retroperitoneal tumors are uncommon neoplastic growths originating in an anatomic area posterior to the abdominal viscera. The purpose of this paper is to report 6 cases of different types of tumors of this region and to review the significant roentgenologic features.

ANATOMY

The retroperitoneal area extends from the diaphragm and twelfth rib to the pelvic diaphragm and is limited laterally by the outer border of the quadratus lumborum muscle. The posterior wall is formed by fascia overlying the vertebrae and the following muscles: psoas, quadratus lumborum and iliacus. Anteriorly the space is bounded by the posterior layer of the parietal peritoneum, part of the posterior surface of the liver, and segments of the colon and duodenum. The retroperitoneal space extends between the peritoneal leaves of the intra-abdominal mesenteries. The normal contents of the retroperitoneal area are: kidneys, adrenals, ureters, pancreas, nervous tissue, lymphoid tissue and major vascular structures which are loosely fixed to each other by fibrous and fatty areolar tissue. Only tumors originating independently of the normal organs or systems in this region are usually classified as primary retroperitoneal tumors. Lymphoid tissue tumors originating within the retroperitoneum are included; however, involvement of this area by generalized dissemination is not considered with these lesions as they present different clinical features.

INCIDENCE AND ORIGIN

Pack and Tabah[7] reported the incidence of primary retroperitoneal tumors to be 0.2 per cent among 60,000 tumor cases. A variety of tissues is found in the retroperitoneal area and tumors originating therefrom are consequently of diversified histologic types and may be benign or malignant. This area is the locale of complex embryologic development of the urogenital apparatus. Investigations by Hansman and Budd[4] indicate that tumors of the retroperitoneum may arise from remnants of the urogenital apparatus. During embryonic development particles of ectoderm may be carried forward with the urogenital apparatus giving origin to dermoids and epithelial cysts. Incomplete degeneration of epithelial structures, extending from the fourth cervical vertebrae to the pelvis during embryologic development, may also give rise to epithelial cysts. Teratomas and solid dermoids are usually considered as originating from totipotent sex cells which were misplaced before the embryo took on definite morphology.

Seventy five to 85 per cent of the tumors have malignant characteristics; of these, the most common are lymphoma, rhabdomyosarcoma, leiomyosarcoma, liposarcoma, and fibrosarcoma. A large group of rare malignant tumors have been identified. The most common benign tumors are cysts although other rare benign lesions have been encountered. The largest series of primary retroperitoneal tumors have been reported by Donnelly,[2] Melicow,[5] Newman and Pinck,[6] Pack and Tabah,[7] and Rabinovitch, *et al.*[9]

Retroperitoneal tumors occur with al most equal frequency in both sexes and are found in all age groups with the greatest rate of occurrence in the fifth and sixth decades. Approximately 60 per cent of the growths occur after the age of forty years, 25 per cent between the ages of ten and forty years, and 15 per cent under the age of ten years. Teratomas have a predisposition for the first decade with 55 per cent occurring under ten years of age and 90 per

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cent before the age of thirty. Approximately one-third of rhabdomyosarcomas are of the embryonal type and occur before the age of ten years.

CLINICAL FEATURES

A retroperitoneal tumor may reach large size before becoming symptomatic as the loose areolar tissue and relative mobility of the anterior wall of the retroperitoneum permit unhampered growth. Symptoms often occur late and are vague and will depend on the organ or system primarily affected by pressure, displacement, or infiltration by the tumor. The most common initial symptoms are abdominal pain, fullness of the abdomen, and gastrointestinal disturbances consisting of nausea, vomiting, and change in bowel habits. Backache and pain or swelling of a lower extremity may occur as the result of pressure by the mass on the nerve roots in the lumbar and sacral region and from impairment of the vascular and lymphatic systems. Dyspnea may result from the tumor displacing the diaphragm upwards. The usual urinary symptoms are frequency, dysuria, and hematuria. Massive retroperitoneal tumor may cause sufficient obstruction of both ureters to produce uremia. Anorexia, weight loss, fever, and weakness occur in advanced malignant growths.

The most frequent physical finding is a painless mass in the abdomen. A retroperitoneal mass in the pelvis may be detected by rectal examination. Ascites, varicosities of the lower extremities, and distention of abdominal veins occur in some cases. A difference in the temperature of the lower extremities from involvement of the sympathetic system by the tumor has been reported. Growth of the neoplasm may extend into the mesentery of the large and small intestine or invade the inguinal canal and produce a mass in the labium or buttock; extension through the obturator foramen may occur. Retroperitoneal tumors are immobile or have minimal motion during respiration.

ROENTGENOLOGIC ASPECTS

Roentgenologic examination will yield positive findings of a retroperitoneal tumor in approximately 60 per cent of the cases. A flat roentgenogram of the abdomen is usually the initial examination and may show characteristic features. A soft tissue mass of palpable size is often visible as an area of different density than the surrounding tissue. A tumor containing a high proportion of fat with a low roentgen-ray absorption coefficient will be visible as a relatively radiolucent area in contrast to surrounding tissue; however, a tumor of high water content or with dense fibrous stroma mixed with the adipose tissue will have an absorption coefficient approaching that of other soft tissue and will not be discernible. A large mass, the density of muscle or fibrous tissue, may be seen as an abnormal area of homogeneous opacity. Calcification within the mass is readily identified and may be diagnostic, as a tooth within a teratoma; amorphous calcification may develop from hemorrhage or necrosis within the mass and then is not specific of a histologic type of tumor. Normal abdominal soft tissue densities may be displaced, distorted, or obscured; these include especially the renal, hepatic, splenic and psoas muscle shadows. The diaphragm may be elevated by a large growth in the upper retroperitoneal area.

Pyelography, intravenous and retrograde, with frontal and lateral projections is usually the most productive method of roentgenologic investigation. A retroperitoneal tumor is in most instances in intimate contact with some part of the urinary system. A kidney may be displaced grossly anteriorly, laterally, or medially across the midline by tumor. Rotation of a kidney on its vertical, horizontal or longitudinal axis by the growth is frequent if the location of the tumor is cephalad. The collecting system usually does not show abnormality unless there are pressure effects producing partial obstruction and causing hydronephrosis. The ureters are displaced when tumors are lower in position and hydro-ureter

may developed from partial obstruction. A retroperitoneal mass in the pelvis produces distortion and displacement of the urinary bladder.

The stomach, small intestine and colon are sometimes displaced or compressed by a retroperitoneal growth. Sheinmel and Mednick[10] demonstrated retrogastric and retroperitoneal tumefactions in the upper abdomen, undetected by the usual positions, by utilizing the left lateral upright and the right lateral recumbent positions. They established normal values for the distance between the spine and reference points of the opacified stomach and duodenum in these positions. The upper abdomen on the left, in the midline, and in the right paravertebral area was rendered more accessible. Figiel and Figiel[3] demonstrated fistulous communication of retroperitoneal neoplasm with the duodenum and jejunum; gas and contrast medium in the gastrointestinal tract partially outlined an irregular necrotic neoplastic cavity.

The diaphragm has decreased motion and is high in position at fluoroscopic examination in bulky tumors of the superior retroperitoneum.

Laminagraphy will aid in further delineation of the tumor and in detection of faint calcifications. Surrounding soft tissue structures are more readily defined.

Retroperitoneal pneumography is an additional procedure that in certain cases aids considerably in diagnosis of tumors in this region and in evaluating the normal organs, especially the adrenal glands. Smaller tumors and lymph nodes, enlarged by retroperitoneal lymphoma, may be visualized only by this procedure. In some situations, as in complete block of the ureter so that pyelography cannot be performed, pneumography may provide the desired information.

Abdominal aortography provides information in selected cases, not obtainable by any other diagnostic roentgenologic procedure. Masses of vascular, extrarenal, and renal origin can be differentiated by this technique. A nephrogram can clarify the relationship of a tumor in juxtaposition to a kidney. Vascularity of a growth is evaluated by aortography. A scanty or absent arterial supply is suggestive of a cystic lesion and of a more favorable prognosis. Pooling of contrast medium within the mass is indicative of its malignant nature.

In particular instances it is advantageous to combine two or more of the above procedures; however, this adds to the risk involved and multiple single examinations are generally preferable.

CASE REPORTS

Case 1. J.R.B., a forty-three year old white male was admitted on November 1, 1948, with progressive abdominal swelling of five months, duration, tiredness, and shortness of breath. Pertinent physical findings were a large nontender mass with irregular contour in the abdomen especially prominent in the right lower and left upper quadrants. The mass could be partially outlined on rectal examination.

Laboratory findings were: hemoglobin of 11.5 grams and a sedimentation rate of 20 mm.; the remaining laboratory studies were noninforming.

Chest roentgenogram showed a high position of the diaphragm, while an abdominal roentgenogram demonstrated a mass of uniform density in the left upper quadrant and nonvisualization of the psoas and renal shadows. Pyelograms, intravenous and retrograde, revealed displacement and moderate dilatation of the right ureter, and minimal blunting of the calyces on the right. The left collecting system was normal. A large central soft tissue mass displaced the colon laterally in all directions on barium enema study; the stomach and duodenum were displaced considerably upward, slightly to the right and slightly posteriorly on examination of the upper gastrointestinal tract (Fig. 1).

On cystoscopic study there was depression of the dome of the urinary bladder by an extravesical mass. A catheter could be passed only half-way into the right ureter.

The tumor did not respond to a moderate dose of radiation therapy and surgical intervention was accomplished on November 24,

Fig. 1. Case 1. Upward displacement of the antrum of the stomach and lateral displacement of the distal part of the duodenal loop and proximal jejunum by retroperitoneal myxosarcoma. The laterally displaced colon is faintly outlined on the left.

1948. There were multiple retroperitoneal masses; one arose with a wide base to the right of the vertebral column, extending from the diaphragm to L-3. A second mass in the pelvis was lying between L-3 and the brim of the pelvis on the right and a third mass, the size of a tennis ball, was attached to the peritoneum in the midabdomen. Biopsy specimens were taken and the histologic diagnosis was myxofibrosarcoma.

There was minimal regression of the masses after postoperative radiation therapy was given to the lower abdomen with 200 kv. peak conventional technique. The patient was readmitted two more times for additional irradiation because of progression of the tumor.

Death occurred on May 7, 1950, and autopsy examination showed the tumor to occupy most of the abdomen with tumor mass weighing approximately twenty-four kilograms and measuring 74 cm. by 46 cm. by 25 cm. Several cystic areas were incorporated within the mass. The myxofibrosarcoma had spread by local extension and by metastatic peritoneal implantation.

Comment. Myxofibrosarcoma is a mixture of fibrosarcoma and myxomatous tissue; the latter may be the result of a degenerative change within the tumor. The behavior of sarcomas containing myxomatous elements is that of the primary histologic tumor type, which in this case is fibrosarcoma. True fibrosarcoma tends to infiltrate slowly and steadily with little tendency to metastasis. The less differentiated tumors show rapid growth.

Case II. H.A.S., a thirty-eight year old white male was first seen on August 4, 1950, for symptoms of a peptic ulcer. Two months before admission he discovered a suprapubic mass but had observed that the lower abdomen had been "hard" for years. He had lost ten pounds of weight during the previous six weeks, apparently from activity of the peptic ulcer; there were no complaints or symptoms referable to the mass.

Physical examination disclosed a very large firm, immovable, low abdominal, midline mass which was not tender. The tumor was palpated rectally and a sigmoidoscope could not be passed further than five inches. Laboratory studies were negative.

A roentgenogram showed a 15 cm. homogeneous density in the lower abdomen and pelvis with a rim of calcification on the superior margin of the mass. The distal halves of the ureters were displaced greatly on intravenous pyelography and there was minimal hydronephrosis on the right. The urinary bladder was markedly elongated and displaced to the left (Fig. 2*A*). There was extrinsic pressure displacing the rectum and sigmoid colon to the left and posteriorly. Loops of distal ileum were displaced away from the pelvis (Fig. 2*B*).

Surgical excision on August 8, 1950, showed the growth to extend from the promontory along the sacrum to the coccyx. Two cystic areas of the tumor were punctured during removal. The mass was rather easily shelled out of a capsule.

Examination of the tumor revealed a well encapsulated fibrous gray and yellow mass containing many cysts filled with bloody fluid. The tumor weighed 577 gm. after evacuation of the cysts. On microscopic examination this was a highly cellular fibroma with recent and old hemorrhage and areas of calcification. This was

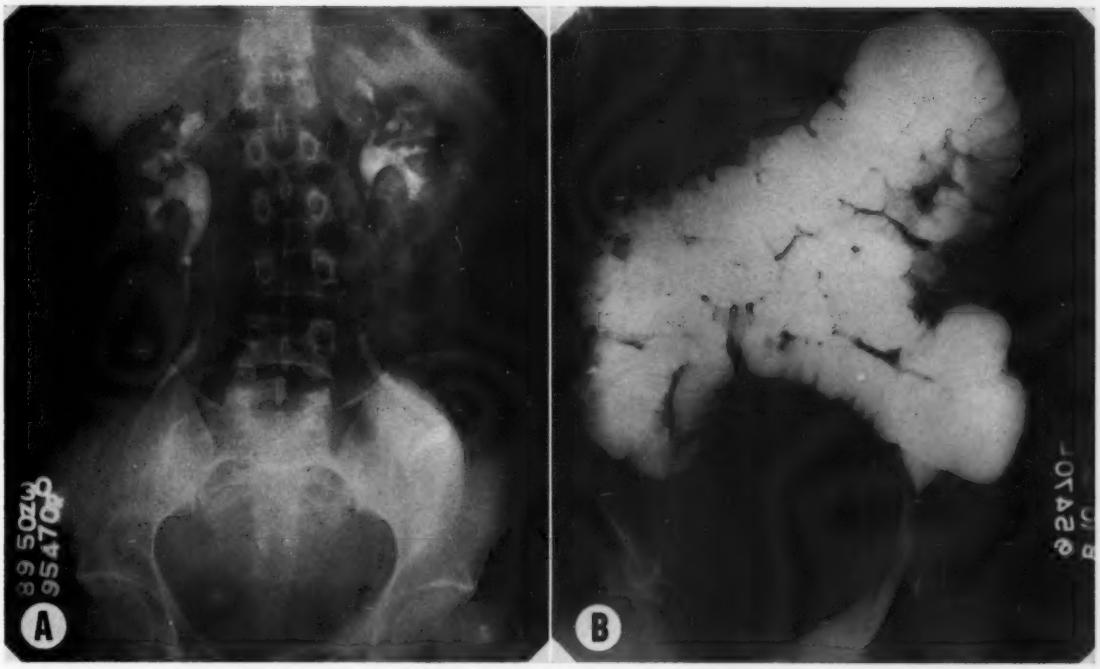

Fig. 2. Case II. (*A*) Lateral displacement of both distal ureters by large retroperitoneal presacral fibroma The faintly visualized urinary bladder is greatly elongated and displaced to the left. Thin curvilinear calcifications are seen on the superior margin of the mass overlying the right sacroiliac joint. (*B*) The barium filled sigmoid colon is displaced to the left, and loops of distal small intestine are pushed away from the pelvis by the fibroma.

considered as having Grade I potentialities of malignancy. On a pyelogram during July, 1953, the urinary system was normal. The last follow-up examination and chest roentgenogram during December, 1954, was normal without evidence of recurrence.

Comment. Fibroma is a benign tumor orginating from fibroblasts while fibrosarcoma is the malignant counterpart. The benign tumor in the retroperitoneal area is less frequently encountered than the malignant growth and produces symptoms by pressure effects upon other organs.

Case III. E.P., a fifty-one year old white male was first admitted during 1948, and again during 1951, for heart disease. He was readmitted during July, 1954, for gradual progressive swelling of the abdomen and a temperature of 100° F. Physical examination showed a chronically ill patient with sallow complexion, a very protuberant abdomen, and a huge ballotable mass on the right; other findings were cardiac fibrillation, ankle edema and ascites.

Pyelography, intravenous and retrograde, revealed displacement of the right kidney and ureter anteriorly and to the left of the lumbar spine by a large homogeneous soft tissue mass in the right flank. There was distortion of the right collecting system (Fig. 3*A*). The small bowel, ascending and transverse portions of the colon were displaced considerably to the left (Fig. 3*B*).

During surgery on August 18, 1954, a soft yellow mass, originating from the region of the right kidney bed and completely enclosed by a thin transparent capsule, was removed from the retroperitoneal area on the right of the spine. There was some extension across the spine. The mass of removed tissue measured 35×25×22 cm. and weighed 5,760 grams (Fig. 3*C*). It was lobulated with a consistency varying from soft to firm. Microscopic examination showed neoplastic adipose tissue and the pathologic diagnosis was liposarcoma.

A pyelogram on January 26, 1955, showed that the right kidney had returned to its usual position; however, there was recurrent fullness in the abdomen and surgical intervention was again performed on February 7, 1955, with incomplete removal of an additional 1,250

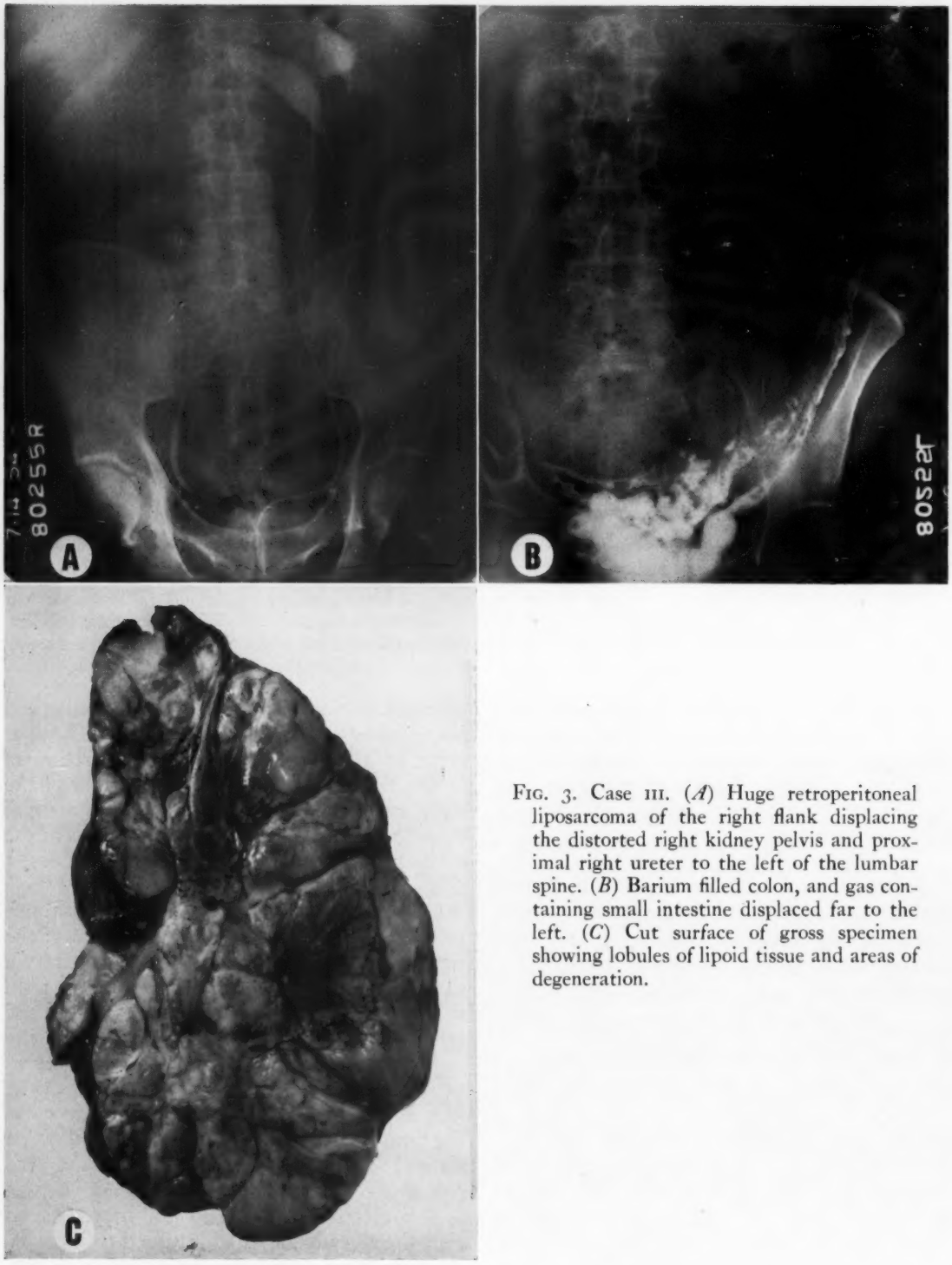

Fig. 3. Case III. (*A*) Huge retroperitoneal liposarcoma of the right flank displacing the distorted right kidney pelvis and proximal right ureter to the left of the lumbar spine. (*B*) Barium filled colon, and gas containing small intestine displaced far to the left. (*C*) Cut surface of gross specimen showing lobules of lipoid tissue and areas of degeneration.

grams of adipose tissue from the right retroperitoneal area. Although microscopic evaluation of the specimen was normal adipose tissue, it was considered as a recurrence of the original liposarcoma.

Comment. Liposarcoma is one of the more common primary retroperitoneal tumors, and occurs oftener in the female sex. It is found in all age groups but has its greatest frequency between the ages of forty and sixty years. This tumor grows slowly and may reach tremendous size. There is an unusual tendency for multiple recurrences. Often the growth is considered benign at the original histologic examination, and sometimes only after several recurrences is it diagnosed as malignant. The tumor probably begins with sarcomatous foci; however, these areas may not be discovered at the original tissue study. Irradiation has a definite place in postoperative treatment and in recurrent, or inoperable liposarcoma since many of these growths are radiosensitive. Liposarcoma usually spreads by local extension or infiltration but occasionally may metastasize.

Case iv. L.B.D., a sixty-two year old white male was admitted during September, 1954, for urinary tract disease with renal tuberculosis diagnosed elsewhere. The chief complaints were urinary frequency and nocturia. He was afebrile.

Physical examination showed the blood pressure to be 258/154. Palpation of the abdomen revealed a firm nontender mass in the right flank. All laboratory studies, including sputum and urine smears and cultures for tuberculosis, were essentially negative.

Roentgenologic studies showed a round, partially calcified mass in the right flank measuring 10 cm. in diameter. An intravenous pyelogram demonstrated upward displacement of the right kidney with rotation on the horizontal axis of the kidney, and minimal medial displacement of the proximal right ureter (Fig. 4*A*). Upper gastrointestinal series and barium enema examination were noninformative.

The right retroperitoneal area was approached through a lumbar incision and a large cystic mass, attached to the peritoneum and adherent to the right kidney, was removed on October 26, 1954.

Grossly the removed kidney was normal in size and appearance except for a few small retention cysts. The cystic mass was attached to the renal capsule only by loose areolar con-

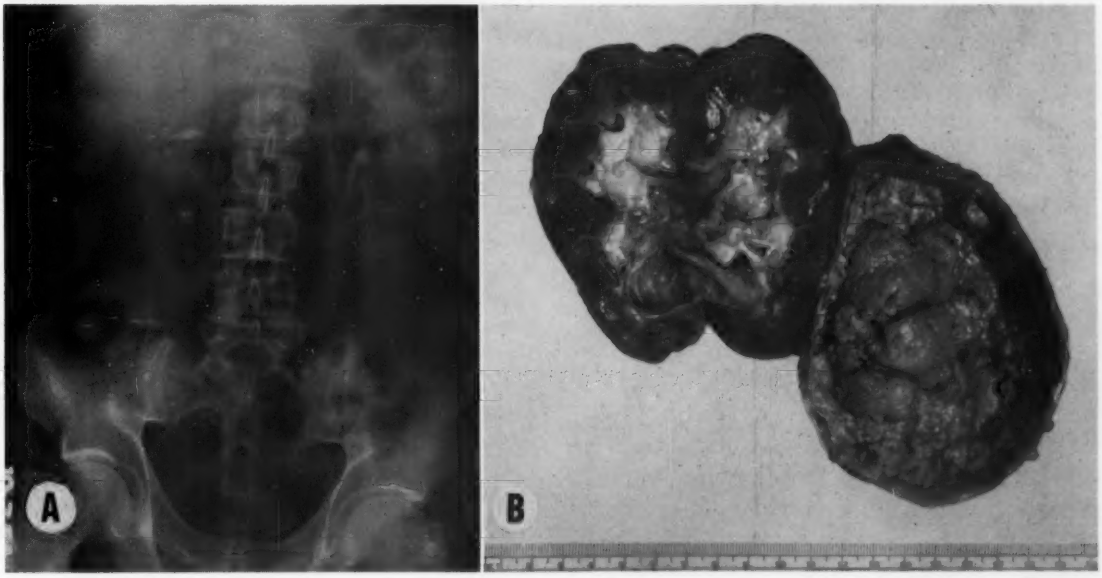

Fig. 4. Case iv. (*A*) Patchy calcified retroperitoneal cyst in the right flank displacing the kidney upward and rotating it on its horizontal axis. The proximal right ureter is displaced slightly medially. (*B*) Gross specimen of cyst loosely attached to capsule of kidney. Nodular masses project from the cyst wall. (Retention cyst in lower pole of kidney.)

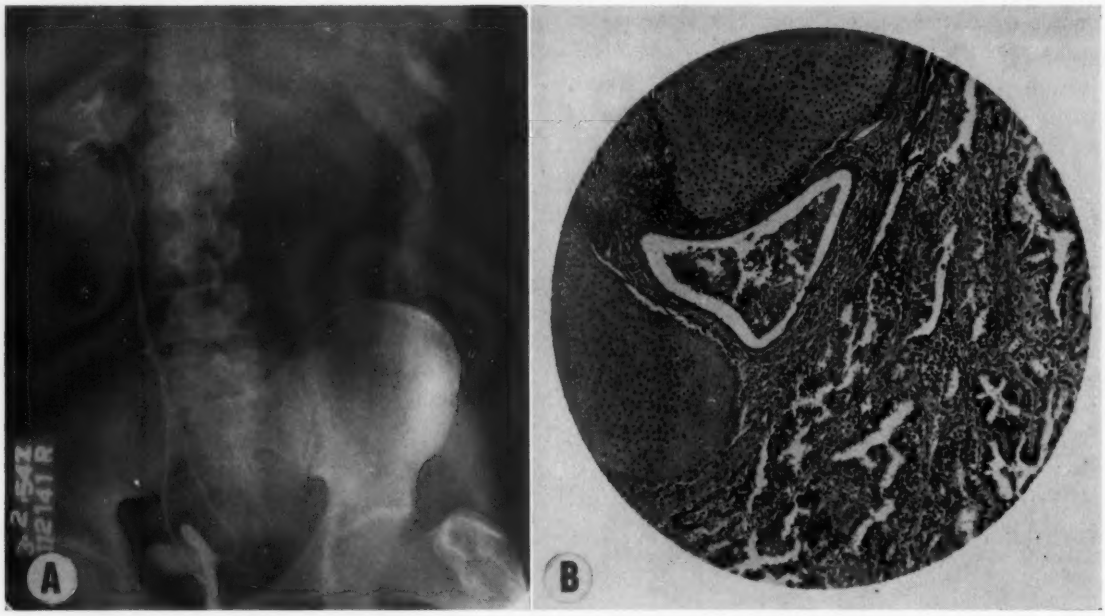

Fig. 5. Case v. (*A*) Primary retroperitoneal teratoma of homogeneous density overlying the sacrum on the left, and displaced distal left ureter medially. The urinary bladder is deviated to the right. (*B*) Photomicrograph of the tumor shows a varied admixture of structures which represents at least two germ layers. There is well differentiated cartilage. A triangular shaped thin walled cyst is lined by low cuboidal epithelium. There are several papillary glandular structures, some lined by ciliated columnar epithelium representative of respiratory tract epithelum; in other parts there are tall columnar cells which are of the mucin secretory type.

nective tissue. The cyst wall was 0.2 cm. thick with a roughened inner surface containing several soft yellowish-brown homogeneous nodules measuring 3 cm. in their greatest diameter (Fig. 4*B*). The cyst was filled with reddish-yellow grumous material. At microscopic examination there was partially calcified, very dense hyaline connective tissue with only amorphous material from the knob-like elevations. The pathologic diagnosis was dermoid cyst.

Comment. The origin of this cyst is obscure. There are several possibilities. A degenerative dermoid cyst in which histologic structures can no longer be identified seems the most probable since there were several nodules in the wall of the cyst and patchy calcification. A cyst may also originate from the embryonal urogenital apparatus or from incomplete degeneration of epithelial structures during embryologic development.

Dermoids and teratomas frequently contain hair and sebaceous material.

Case v. J.V.H., a thirty-four year old white male was examined during December, 1953, for sudden pain in the left lower extremity. Seven years before he had a series of bilateral vein ligations for varicosities of the lower extremities. There were no abdominal symptoms.

During physical examination a hard, nontender, four inch spherical mass was found in the left lower quadrant. Laboratory findings were normal.

A large homogeneous density in the pelvis on the left, which displaced the distal left ureter and the distorted urinary bladder to the right of the midline was revealed by intravenous and retrograde pyelography (Fig. 5*A*). Barium enema examination showed the sigmoid colon to be displaced to the right and posteriorly by the mass.

Surgical exploration on March 4, 1954, disclosed a retroperitoneal tumefaction in the left iliac fossa which originated in the left psoas muscle and extended inferiorly to the level of the lower sacrum. It was necessary to split the psoas muscle to reach the tumor. During removal of the tumor approximately 300 cc. of dark brown serous fluid spilled from a cystic compartment.

According to the pathologist's report, the mass measured 10 cm. by 15 cm. and was multiloculated. Many microscopic sections were examined and there were mingled tissue elements in varying stages of differentiation. There were numerous islands of immature and well formed cartilage. Tubular structures of various size and shapes were another prominent component; these were lined by cuboidal or columnar epithelial cells which often had had mucous secretion and occasionally were ciliated. In addition there were masses of large undifferentiated cells with hyperchromatic nuclei and very irregular shape. In scattered areas the tumor cells formed vascular channels. At one point a small cyst was filled with keratinized material suggesting squamous epithelial elements. A diagnosis of malignant teratoma was made (Fig. 5*B*).

A small spermatocele, confirmed by histologic study, was removed from the left testicle shortly thereafter. The left testicle was incised in several places and was normal; the right testicle was negative. The course of the patient subsequently was progressively poor with local recurrence and metastases to the para-aortic lymph nodes, determined at surgery of May 11, 1954. The recurrent tumor was densely adherent to the femoral vessels. There was intractable pain necessitating chordotomy. Several severe hemorrhages resulted from tumor invasion and necrosis; death occurred on November 5, 1954. Autopsy showed extensive abdominal and retroperitoneal metastases. The testicles were negative for tumor.

Comment. The retroperitoneal and sacrococcygeal areas are the third most common site of teratoma, with the ovaries and testicles being the most frequent location. Teratomas are the third most common retroperitoneal tumors in children ranking in frequency after neuroblastoma and Wilms' tumor; they constitute 10 per cent of primary retroperitoneal tumors of all ages. These tumors occur in all age groups, but predominantly during infancy and childhood.

The tumor probably arises from abnormal tissue primordium in embryonic life which undergoes unregulated differentiation into various tissues. The growth is usually in a paramedian location. There is malignant degeneration in approximately 10 per cent. Teratomas in the retroperitoneal area have been reviewed by Arnheim[1] and Palumbo, *et al.*[8]

Case VI. E.O.F., a fifty-five year old white male, was admitted to a hospital, during December, 1950, with vague gastrointestinal complaints and tenderness in the right upper quadrant.

Multiple roentgen examinations were negative. A subsequent intravenous pyelogram showed nonfunction of the left kidney and retrograde studies demonstrated hydronephrosis of this kidney.

On July 25, 1951, an exploratory operation was performed for unexplained ascites. There was a rather large retroperitoneal mass near the vena cava adjacent to the kidney and a biopsy specimen was taken; histologic examination was interpreted as malignant lymphoma of the reticulum cell type.

Radiation therapy was administered. Mental disturbances developed on September 25, 1952, and he expired on October 10, 1952. Autopsy showed invasion and obstruction of the ureter. Lymph nodes up to 4 cm. in diameter were found matted together to form a mass extending from the celiac nodes to the bifurcation of the aorta and about 10 cm. laterally from the midline. The lumbar vertebrae and brain were also invaded by reticulum cell sarcoma

Comment. Primary lymphoma of the retroperitoneum is the most frequent of all types of primary tumor in this area; lymphosarcoma, including reticulum cell, giant follicular cell and lymphatic lymphoma, occurs from three to seven times as often as Hodgkin's Disease. Lymphomas are usually considered as multicentric in origin; however, certain cases have a course suggesting unifocal origin and may be cured by extirpation if accomplished before lymphatic or hematogenous dissemination has occurred. Complete surgical removal is seldom possible with lymphomas in the retroperitoneum as the tumor is not diagnosed until it has been active for some time and has either attained considerable size or has infiltrated major structures. Symptoms of fever, weakness and weight loss are encountered more often with this type of re-

troperitoneal growth. Radiation therapy and nitrogen mustard frequently offer very gratifying palliation and definitely prolong the useful life of the patient.

TREATMENT

The primary definitive treatment of tumors of unicentric origin is total extirpation if possible; however, this is frequently impossible as invasion of, or proximity to, vital organs and major blood vessels precludes removal of tumor with an adequate margin of normal tissue. Lymphomas, tumors of multicentric origin, are preferably treated by irradiation. Incomplete removal, while not curative, occasionally results in striking palliation on a mechanical basis of removal of bulky neoplastic tissue causing pressure effects on major organs and structure. The majority of retroperitoneal tumors are radiosensitive and will show some degree of response.

The following are the indications for radiation therapy: inoperable tumors; recurrence following resection; incompletely removed tumor; multicentric origin, and as an adjuvant to surgery. Occasionally irradiation administered prior to operation, and after diagnosis has been established, will facilitate the surgical procedure. Supervoltage radiation therapy has an advantage over conventional deep roentgen-ray techniques in these deep seated tumors. Neurosurgical silver clips applied at the time of surgery will aid in directing and localizing the beam of radiant energy in the event of subsequent roentgen therapy.

The prognosis depends upon the type, location and extent of the tumor. Recurrences after surgery appear in 35 to 50 per cent of the cases and show increasing malignancy each time.

CONCLUSIONS

1. Six cases of primary retroperitoneal tumors are reported and pertinent aspects of tumors in this region are discussed.

2. Roentgenologic examination is the most productive and informative nonoperative method of investigation of the retroperitoneum.

3. The roentgenologic features of primary tumors of the retroperitoneum are reviewed.

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The author wishes to express his appreciation to Dr. James E. Loftstrom.

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THE CLINICAL USE OF PITRESSIN IN EXCRETORY UROGRAPHY*

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TWO necessary conditions for good diagnostic results in excretory urography are a normal rate of excretion of the contrast material by the kidneys and an appropriate concentration in the major collecting systems. The first is dependent on renal hemodynamics and a sufficient number of normally functioning nephrons. The second condition is also dependent on normal renal function but, in addition, demands the action of the antidiuretic hormone (ADH), vasopressin. When there is a marked pathologic reduction in the number of functioning nephrons, an adequate excretory urogram cannot be obtained. If, however, the contrast material is excreted at a normal rate, but inadequately concentrated because of diminished ADH activity, as occurs with hydration, the administration of pitressin may result in a diagnostic urographic examination. The present study was undertaken to determine whether or not diagnostically adequate excretory urograms could be routinely obtained in patients who were not prepared by a dehydration regimen, but who were given pitressin intramuscularly.

PHYSIOLOGY

The antidiuretic hormone is thought by many (Rothballer and Dugger,[11] Scharrer and Scharrer[12]) to be secreted in the supraoptic and paraventricular nuclei located in the anterior portion of the hypothalamus and then stored in the posterior lobe of the pituitary gland. The secretion of ADH is responsive to several stimuli, but Verney[14] has shown that the most important of these is an increase in the effective osmotic pressure of the fluids bathing a receptor mechanism in the anterior portion of the hypothalamus.

In the kidney the osmotic pressure (molecular concentration) of the glomerular filtrate is essentially identical with that of plasma. The total filtrates amount to approximately 180 liters per 24-hour period and are reduced to 1.5 liters of bladder urine (Fig. 1). It is believed that 85 per cent of the reduction occurs in the proximal tubule due to an active reabsorption of solutes and the associated obligatory passive diffusion of water. Thus, only 15 per cent of the total volume, altered in composition, but still isosmotic with the filtrate, reaches the distal tubule. Further active reabsorption of solutes from this small volume of fluid occurs in the distal tubule but, unlike the process in the proximal

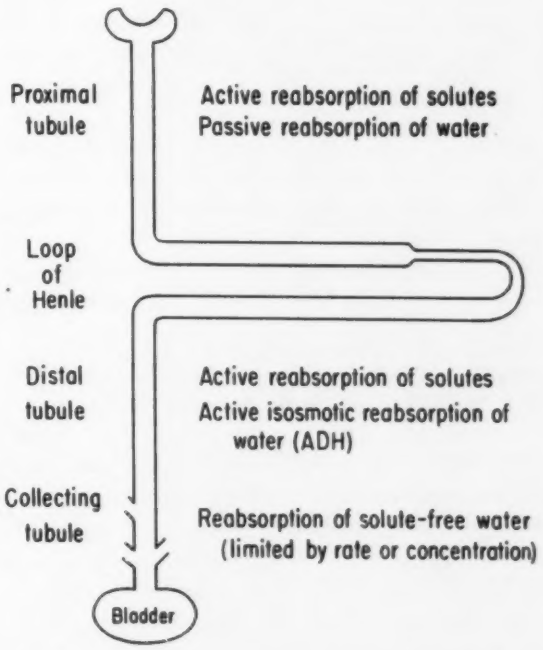

FIG. 1. Schematic drawing showing the sites of passive and active reabsorption of water.

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tubule, does not necessitate the passive diffusion of water (Smith,[13] Welt[15]). Rather, the reabsorption of water requires the action of ADH. The exact mechanism of the hormonal action is not known (O'Connor[10]).

With maximal ADH action in the distal tubule, a small volume of isosmotic fluid reaches the collecting tubule where water may be further reabsorbed independent of solutes. The absorption by the collecting tubule has two limiting parameters, namely, a rate of 5–7 cc. per minute and an osmotic gradient such that concentration cannot exceed 1,400 milliosmols per liter (Fig. 1). Therefore, it is apparent that in the absence of ADH action in the distal tubule the large volume of dilute fluid reaching the collecting tubule cannot be transformed to concentrated urine, even when water is abstracted at the maximal rate.

In ordinary roentgenologic practice secretion of pitressin is promoted by asking the patient to refrain from drinking fluids for about 12 to 18 hours. During this interval the patient continues to lose fluid as insensible and sensible perspiration, breath and urine; hence the body fluids become more concentrated and the osmo-receptor is stimulated. Dehydration has several disadvantages: The undesirability of withholding needed fluids in certain patients; the occasional need for the examination in an emergency situation; the discomfort of thirst for the patient; and the risk that preparatory orders will not be carried out.

MATERIAL

As pitressin has effects on other organs (Freidman and Pauls,[2] Kordik,[7] and Whittlesey[18]), it was administered only to hospitalized adult patients, which allowed a better opportunity to reject those in whom its use might be harmful. Patients with hypertension and/or coronary artery disease were not given pitressin. Pregnant women also were excluded. The non-pitressin group comprised all adult outpatients referred for urography except those with hypertension and/or coronary artery disease more severe than grade 1. Thus the two groups were comparable. Five hundred consecutive urographic examinations were performed and the roentgenograms classified by four radiologists as excellent, good, fair or poor.

The criteria were similar to those used by Mullen, *et al.*,[9] and were as follows:

1. Excellent: Urogram with diagnostic quality approaching a retrograde pyelogram.

2. Good: Urogram with satisfactory demonstration of all portions of the urinary tract so that a diagnosis could be made without recourse to a retrograde study.

3. Fair: Urogram showing only a portion of the urinary tract satisfactorily; the need for a retrograde study indicated by the clinical problem.

4. Poor: Urogram non-diagnostic; further study needed.

During the study a separate record was kept for each patient. After elimination of cases because of insufficient data regarding fluid and food intake, or other pertinent information about the examination, 227 of the pitressin group and 226 of the non-pitressin group remained for analysis. The classification of the examinations is shown in Table 1 and the reactions that occurred are given in Table II. No reaction attributable to pitressin occurred.

PROCEDURE

The pitressin group received no preparation; fluid and food were ingested as usual. Since the aqueous solution of pitressin has some antidiuretic effect during a period of

TABLE I

CLASSIFICATION OF EXAMINATIONS

Classification of Examination	Pitressin Group	Percentage	Non-Pitressin Group	Percentage
Poor	26	11.5	24	10.6
Fair	53	23.3	79	35.0
Good	112	49.3	93	41.1
Excellent	36	15.9	30	13.3
	227	100.0	226	100.0

TABLE II

TABULATION OF REACTIONS

	Nausea	Emesis	Arm Pain	Flushing (severe)	Other
Pitressin	35	21	7	2	6
Non-Pitressin	53	26	6	2	6

two hours (Wilson and McGinty[19]) one unit of aqueous pitressin* was injected intramuscularly 35 to 40 minutes before the intravenous administration of 25 cc. of 50 per cent urokon.† The non-pitressin group was prepared by the usual restriction of food and fluids for 12 to 18 hours prior to the examination.

Each patient was questioned concerning an allergic diathesis. When a positive history was obtained, a 50 mg. dose of pyribenzamine‡ was given orally and the examination deferred for 30 minutes. Skin or eye sensitivity tests (Archer and Harris[1]) were not used, but after the intravenous injection of 1 cc. of the contrast material there was a one- to two-minute delay to be certain there was no reaction before the remainder was injected. Compression of the lower abdomen was used routinely (Harrow[4]). The examination included a survey roentgenogram and pyelograms at 5, 10 and 15 minutes after the injection of the contrast medium. Special additional views were dictated by the findings noted on the 5-minute pyelogram which was always inspected immediately after processing (Keates[5,6]).

STATISTICAL ANALYSIS

The results of the classification and the incidence of reactions in the two groups were analyzed statistically.

Tests of Statistical Significance

A. A rating scale was used for all the examinations. The scores were: poor film, 0; fair film, 1; good film, 2; excellent film, 3.

Using this rating scale the following was calculated:

Number of Films		*Average Grade*	*Standard Deviation*
227	pitressin	1.696	.8725
226	non-pitressin	1.571	.8520

$$t = \frac{\bar{x}_1 - \bar{x}_2}{S_{\bar{x}_1 - \bar{x}_2}} = 1.54 \text{ with } 451 \text{ degrees of freedom.}$$

The probability of such a value of t is not significant.

Conclusion: The average grade for the pitressin group does not differ significantly from the average grade for the non-pitressin group. Both groups gave a fair to good examination on the average though the pitressin group was slightly better.

B. Comparison of percentage of *poor+fair* films from pitressin group to non-pitressin group

Number of Films		*Percentage Poor+Fair*
227	pitressin	34.8
226	non-pitressin	45.6

$$z = \frac{p_1 - p_2}{\sqrt{\frac{p_1 q_1}{N_1} + \frac{p_2 q_2}{N_2}}} = 2.36.$$

The probability of such a value of z is approximately 2.5 per cent.

Conclusion: There is a real difference in percentages of poor+fair films for the two groups unless a small chance (less than 3 in 100) has occurred in random sampling. Thus, a significantly higher percentage of re-examinations is necessary in the non-pitressin group.

C. Test of the association of a reaction with pitressin

	Little or No Reaction	*Mild Reaction*	*Serious Reaction*	
pitressin	178 (170.88)	47 (54.12)	2 (2.00)	227
non-pitressin	163 (170.12)	61 (53.88)	2 (2.00)	226
	341	108	4	453

The figures in ( ) are expected values.

$$\chi^2 = \sum_{\substack{\text{over} \\ \text{all} \\ \text{classes}}} \frac{(\text{observed-expected})^2}{\text{expected}} \text{ with 2 degrees of freedom.}$$

$\chi^2 = 2.47$ with 2 degrees of freedom.

The probability of such a value of χ^2 is not significant.

* Parke, Davis and Co., 20 units diluted with distilled water (1 unit per ½ cc.)

† Mallinckrodt Chemical Works.

‡ Ciba Pharmaceutical Products, Inc.

Conclusions on the basis of all tests: The diagnostic quality of the roentgenograms does not appear to differ markedly whether pitressin or dehydration is used, though a higher percentage of examinations in the non-pitressin group was rated poor or fair. Assuming that all fair and poor examinations would require either retrograde or repeat excretory urograms to establish a diagnosis, the use of pitressin significantly reduces the number of patients subjected to these additional studies. More reactions of a mild or serious nature were recorded in the non-pitressin group (some reactions in 28 per cent of patients) than in the pitressin group (some reactions in 22 per cent of patients). The data give no evidence of an association between the use of pitressin and a reaction.

DISCUSSION

In his paragraph on preparation of the patient, Wesson[17] recommends the use of two doses of pitressin (0.5 cc. each) which would be equivalent to 10 units of USP pitressin, given intramuscularly to decrease the amount of colonic air. No significant decrease in the amount of this interfering element was noted in our pitressin group, probably because of the small dose that was used. The dose of pitressin (one unit) is considered more than adequate to obtain the desired renal action (Maxwell and Breed[8]), and it is likely that a smaller dose would be adequate (Welt[16]). However, this dose of ADH is much less than the 5 to 20 units needed to elicit the response on the smooth muscle of the intestine and incalculably small when compared to the amount of ADH required to produce a vascular reponse (Goodman and Gilman[3]).

The evaluation of these studies shows that in the pitressin group the ureters were repeatedly opacified more completely and the amount of contrast material in the bladder 10 minutes after the injection was less than in the non-pitressin group and resulted in a roentgenogram on which the outlines of the calyces, pelves and ureters approached those observed in a retrograde examination. This is thought to be due to the decrease in total urine volume secondary to the ADH activity since the ureter is not known to be affected by pitressin.

The use of the antidiuretic hormone in excretory urography has proved of value in various clinical conditions. The usual maintenance dose of pitressin for patients with diabetes insipidus was simply augmented to obtain diagnostic urograms. For patients with diabetes mellitus, the restriction of fluids and interference with insulin dosage can be obviated during intravenous pyelography. Diagnostic examinations can be performed on surgical patients while they are receiving intravenous fluids in the immediate postoperative hours. Without the need for a period of dehydration, uncooperative and mentally incompetent patients can be more easily studied. If no contraindications are present, pitressin can be used in "emergency" excretory urography, including accident cases. Occasionally, orders for dehydration may be omitted and by the use of pitressin the patient examined satisfactorily without prolongation of his hospitalization.

The clinical selection of patients for the administration of pitressin is not a great disadvantage, and may be unnecessary with the use of a smaller dose. The slight increase (35 minutes) in the length of the patient's stay in the radiologic department and the possible hazard of aspiration of food particles in those instances of emesis secondary to the administration of contrast material also are considered minor disadvantages.

SUMMARY

A series of 453 excretory urograms was analyzed to determine whether or not the intramuscular injection of one unit of pitressin to hydrated patients would result in examinations that were comparable to the studies obtained in patients who were dehydrated.

Statistical analysis discloses that the examinations in the pitressin group were equal in quality to the studies in the non-

pitressin group and, in addition, fewer re-examinations are necessary. Pitressin is of value in "emergency" urography and in several clinical conditions in which dehydration is undesirable. No serious disadvantage has been encountered.

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Representative on American Board of Radiology: H. D. Kerr, St. Michaels, Md., T. Leucutia, Detroit, Mich.

Director of Instructional Courses: H. O. Peterson, St. Paul, Minn., *Manager of Annual Meeting:* James C. Cook, 110 Professional Building, Detroit 1, Mich.

Editor: Lawrence Reynolds, 110 Professional Building, Detroit 1, Mich.

Fifty-eighth Annual Meeting: Shoreham Hotel, Washington, D. C., October 1–4, 1957.

AMERICAN RADIUM SOCIETY

President: Ralph T. Ogden, Hartford, Conn.; *President-Elect:* Milford D. Schulz, Boston, Mass.; *1st Vice-President:* Ralph M. Caulk, Washington, D. C.; *2nd Vice-President:* Elizabeth F. Focht, New York, N. Y.; *Secretary:* Theodore R. Miller, 139 East 36th St., New York 16, N. Y.; *Treasurer:* Jesshill Love, St. Joseph Infirmary, Louisville, Ky.

Executive Committee: John E. Wirth, Chairman, Pasadena, Calif.; Grant H. Beckstrand, Long Beach, Calif.; Norman A. McCormick, Windsor, Ontario, Canada.

Program Committee: Ralph T. Ogden, Chairman, Hartford, Conn.; Franklin L. Payne, Philadelphia, Pa.; William S. MacComb, Houston, Texas; John L. Pool, New York, N. Y.; Donald S. Childs, Jr., Rochester, Minn.; Edith H. Quimby, New York, N. Y.; Milford D. Schulz (ex officio), Boston, Mass.

Publication Committee: Harry Hauser, Chairman, Cleveland, Ohio; Martin Van Herik, Rochester, Minn.; J. Robert Andrews, Bethesda, Md.

Public Relations Committee: J. Samuel Binkley, Chairman, Oklahoma City, Okla.; Ernest H. Wood, Chapel Hill, N. C.; Frederick W. O'Brien, Jr., Worcester, Mass.

Janeway Lecture Committee: Gioacchino Failla, Chairman, New York, N. Y.; H. Dabney Kerr, St. Michaels, Md.; Lloyd F. Craver, New York, N. Y.

Representatives on the American Board of Radiology: Douglas Quick, New York, N. Y.; B. P. Widmann, Philadelphia, Pa.; Frederick W. O'Brien, Boston, Mass.

Representatives on National Committee on Radiation Protection: Edith H. Quimby, New York, N. Y.; Theodore P. Eberhard, Ann Arbor, Mich.

Representative on the Board of Chancellors of the American College of Radiology: Charles L. Martin, Dallas, Texas.

Committee on Arrangements: Robert L. Brown, Chairman, Emory University, Ga.; Jesshill Love, Vice-Chairman, Louisville, Ky.; Wade S. Rizk, Jacksonville, Fla; Herbert D. Kerman, Daytona Beach, Fla.; J. Samuel Binkley, Oklahoma City, Okla.; Ralph T. Ogden, Hartford, Conn.

Fortieth Annual Meeting: Hollywood Beach Hotel, Hollywood Beach, Florida, March 27–29, 1958.

EDITORIALS

THE ANNUAL MEETING OF THE AMERICAN RADIUM SOCIETY

THE American Radium Society held its Thirty-ninth Annual Meeting in Quebec, Canada, on May 29, 30, 31 and June 1, 1957 at the charming Chateau Frontenac. The total number registered was 321, which was the largest attendance on record, comprising 91 members, 111 guests and 119 ladies and children.

On Wednesday evening, May 29, a reception was held for the members and guests by the President and Members of the Canadian Association of Radiologists in the beautiful Salon Champlain of the Chateau. The next morning the meeting was officially called to order by Dr. Norman McCormick of Windsor, Ontario, who delivered the Presidential Address. The theme of his subject was the development and activities of the Ontario Cancer Foundation, which was organized in 1932 by the Provincial Legislature to investigate the use of radium and to prepare a plan for the satisfactory dispersal of a considerable amount of public monies allocated for this purpose. Later the Foundation established properly located clinics in order to conduct a program of diagnosis, treatment and research in cancer. After completing his address, Dr. McCormick asked the audience to stand in memory of Dr. Douglas J. Roberts of Hartford, who died unexpectedly and suddenly on March 12, 1957. A summary of Dr. Roberts' professional activities was read by the President. The program continued with the presentation of the Gordon E. Richards Memorial Lecture by Dr. Arthur W. Ham of Toronto, who spoke on the newer knowledge of the structure of normal and malignant cells obtained by electron microscopy. Three other papers were presented during this session, including a discussion of high resolution x-ray microscopy, end results of treatment of cancer of the hypopharynx, carcinoma of the larynx and electrolyte balance studies in the postradiation state. In discussing cancer of the larynx, Drs. William S. MacComb and Gilbert H. Fletcher of Houston gave the indications for irradiation and surgery and stressed the importance of combined therapy in some patients.

In the afternoon scientific session a Round Table Conference on *Treatment of Primary Carcinoma of the Lung* was moderated by Dr. Douglas Quick. Some of the subjects discussed were the influence of pathology on the choice of treatment, physical factors in radiation therapy of the lung, five-year survivals in surgical treatment, interstitial implantation with radioactive gold in nonresectable lesions, combined roentgen and nitrogen mustard therapy and supervoltage irradiation of inoperable lung cancer. Later in the afternoon an interesting paper on the effect of Co^{60} beam therapy was given by Drs. Ivan H. Smith and Stewart Lott in which they pointed out the advantages of decreased bone absorption, lessened integral dose and increased depth dose in treating oral, esophageal, laryngeal and bladder carcinomas. The final paper of this session which was given by Dr. Robert A. Mustard of Toronto was on the surgical management of malignant tumors of the salivary glands.

In the early evening, a *Vin d'honneur* for members and guests of the American Radium Society was tendered by L'Université Laval. Monseigneur Alphonse Parent, Recteur, proved a gracious host to the group in

the attractive Salon de l'Université. Following this function, the group proceeded by bus to *Manoir Saint Castin* at *Lac Beauport* for an informal dinner. The setting at this lodge at the foothills of the Laurentians was most attractive and restful. Following dinner, Dr. Carleton B. Peirce introduced the guest speaker of the evening, Dr. George C. Laurence, Director of Research and Development, Atomic Energy of Canada, Ltd. Dr. Laurence spoke on the present and future peaceful applications of atomic energy. He also explained the use of uranium as a fuel in the production of electric power.

The second day of scientific sessions began with a symposium on rotational therapy. The various subjects discussed were: the history of rotational therapy, the physical aspects, rotational therapy at 250 kv., supervoltage rotation with a van de Graaff generator, supervoltage rotation irradiation of carcinoma of the head and neck, horizontal rotational therapy with Co^{60}, and clinical experience with the theratron. A preliminary report on clinical betatron therapy with the consideration of the applicability of rotation was also presented. A lively round table discussion of the papers on the symposium was carried out by the essayists.

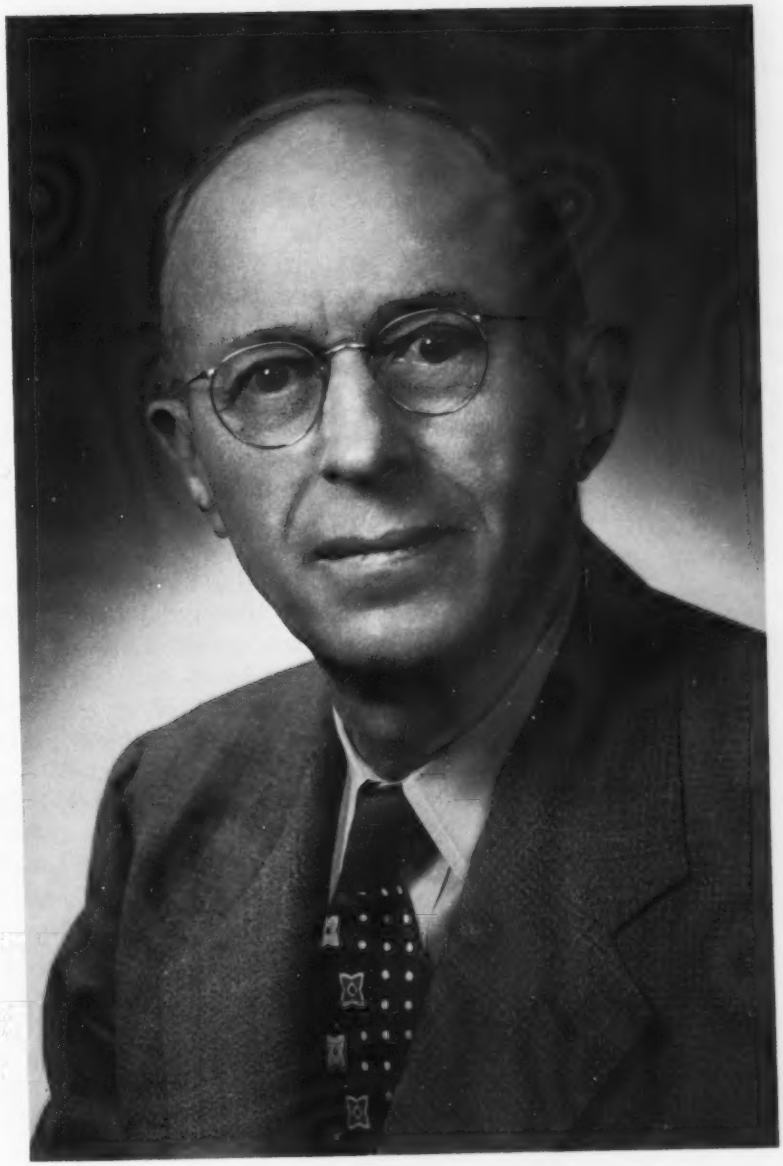
The afternoon session was opened by Drs. Jean Bouchard and Carleton B. Peirce, who presented an interesting communication on the use of different energy levels of ionizing radiation required for therapeutic radiology. Dr. Edith H. Quimby gave a practical report on the training requirements and training courses for the medical use of radioactive isotopes. A variety of other subjects was presented, including the use of interstitial radium, dose-rate tables for the radioactive isotopes, effects of irradiation on metastatic lymph nodes from various primary carcinomas and the therapeutic value of whole-body roentgen irradiation for patients with generalized neoplastic disease. Following a short intermission, Dr. Gioacchino Failla introduced Dr. Simeon T. Cantril, who delivered the Janeway Lecture. He spoke on *Contributions of Biology to Radiation Therapy*. He reviewed in great detail the history and literature relating to biologic experimentation and clinical experience by workers since the beginning of the medical use of ionizing radiations. He emphasized that much of the information gained early in the century has proved to be of great value in present-day application in the treatment of neoplasms. He also pointed out the responsibility of our profession of being fully informed concerning the possible injury which may accrue from environmental contamination by military and peaceful application of nuclear energy. He closed his address by quoting from Regaud and Lacassagne: "Les progrés de nos connaissances sur les causes de la radiosensibilité ont été lents, notre ignorance sur ce sujet est encore grande."

On Friday evening the Radium Chemical Company of New York City sponsored a social hour in the Salon Champlain of the hotel. Following this entertainment, the Annual Banquet was held in the ballroom of The Chateau. Dr. Norman McCormick presented the Janeway Medal to Dr. Cantril after reciting the legend of Odin. Dr. Cantril expressed his appeciation for this great honor and paid tribute to his associates, past and present.

The scientific session on the closing day included a variety of contributions.

The final social event of the meeting was a luncheon for the members and guests in the ballroom of The Chateau, sponsored by the Atomic Energy of Canada, Ltd. The President of the Society expressed his deep appreciation for the excellence of the work performed by the Committee on Arrangements and the Ladies' Hospitality Committee. He also expressed his appreciation to the Canadian members and their wives for their cordiality, and to the various organizations sponsoring entertainment.

Harry Hauser, M.D.



JOHN HEBERDING
1882-1957

DR. JOHN HEBERDING died on January 26, 1957. He was known as the "Father of X-ray" in Youngstown, Ohio. He was born November 17, 1882 in Struthers, Ohio, a son of Joseph and Charlotte Heberding.

"Dr. John" graduated from Western Pennsylvania Medical School (now University of Pittsburgh) in 1905. He served his internship at Youngstown Hospital Association (South Side Unit). It was here that he first came in contact with the then "new x-ray machines."

After a few months of combined general

practice and x-ray work, Dr. Heberding took some postgraduate study under the late Dr. George Pfahler of Philadelphia. He continued the combined practice for about twelve years, after which he began to specialize in roentgenography.

Dr. John was the Chief of the radiology department of Youngstown Hospital Association. Upon his retirement as Chief emeritus, he continued active directorship of the radiology department, North Unit of Youngstown Hospital Association and also maintained his private practice.

In April, 1955, Dr. John was the first to make the diagnosis of his illness—carcinoma of the colon. Following surgery, he was able to devote about twelve months to his first love—radiology. Then after his own diagnosis of metastases to the liver, he was forced by weakness to retire.

Throughout his entire illness and especially the last few months, I was privileged to witness a perfect example of the doctor, the man, and the person who had lived a full life and was not afraid of the future. He described his own illness as objectively and without emotion as if he were talking of some other case.

Dr. John was always a student of medicine in general, and radiology in particular. The newer methods and developments were always explored by him and put into use, if found practical.

In 1955, Dr. Heberding received his "Fifty Year" pin (practicing for fifty years) from the Ohio State Medical Society. He was a member of the Rotary Club, Western Star Lodge, F.&A.M., Knights Templars, Methodist Church, Mahoning county, Ohio State and American Medical Associations, Cleveland Radiological Society (past president), Ohio State Radiological Society, and of the American Roentgen Ray Society. He was a Fellow of the American College of Radiology and a Diplomate of the American Board of Radiology.

He is survived by his widow, two sons, two daughters and five grandchildren.

Youngstown has lost a beloved practioner of medicine, an excellent radiologist, and a friend of all. American radiology has lost one of its pioneers.

Ben S. Brown, M.D.

SOCIETY PROCEEDINGS AND NEWS ITEMS

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY

The Nineteenth Midsummer Radiological Conference of the Rocky Mountain Radiological Society will be held on August 15, 16 and 17, 1957, at the Shirley-Savoy Hotel, Denver, Colorado.

The guest speakers are (in order of appearance): Benjamin H. Orndoff, M.D., Paul C. Abersold, Ph.D., Titus C. Evans, Ph.D., Harry M. Spence, M.D., Russell H. Morgan, M.D., and Henry L. Jaffe, M.D.

An excellent program has been arranged. For further information, please contact Dr. John S. Bouslog, President, 304 Republic Building, Denver 2, Colorado.

ORNL AND ORINS SPONSOR SYMPOSIUM

A symposium on high-energy physics featuring outstanding scientists in the field will be held in Oak Ridge, Tennessee, August 19–24, 1957. The symposium will be presented under the joint sponsorship of Oak Ridge National Laboratory and the Oak Ridge Institute of Nuclear Studies.

The six-day program will be conducted on an introductory level and will be addressed to scientists who have not previously been working in the field of high-energy, or elementary-particle, physics.

The lecturers and conference leaders scheduled to take part in the symposium are Dr. Maurice Neuman and Professor L. Alvarez, University of California; Professor Martin Block, Duke University; G. T. Zorn, Brookhaven National Laboratory; Professor A. Pevsner, Johns Hopkins University; Dr. Val L. Fitch, Princeton University; and Professor D. T. King, University of Tennessee.

Further information about the symposium may be obtained by writing the University Relations Division, Oak Ridge Institute of Nuclear Studies, Post Office Box 117, Oak Ridge, Tennessee.

AMERICAN BOARD OF RADIOLOGY ELECTS OFFICERS

The following officers were elected at the meeting of the American Board of Radiology in Tampa, Florida, April 1–6, 1957: Donald S. Childs, President; Bernard P. Widmann, Vice-President; H. Dabney Kerr, Secretary; and James W. J. Carpender, Treasurer.

POSTGRADUATE COURSE IN RADIOLOGY TO BE PRESENTED

The Postgraduate Division of the School of Medicine of the University of Southern California will present a postgraduate course in "The Physics of Diagnostic and Therapeutic Radiology" from September 6, 1957 to January 24, 1958. Twenty lectures will be given on Friday afternoons from 4:00 to 5:00 P.M. by The Department of Radiology at the Los Angeles County Hospital. The course is designed for physicians preparing for the specialty of radiology. Those physicians who are preparing for the specialty of dermatology will also benefit from the course. Lectures will cover fundamental x-ray physics, determination of quality and quantity of radiation, tissue dosage from x-rays, radium and radon, radiation protection and radiation biology. Tuition, $50.00 for twenty weeks.

For further information, contact Phil R. Manning, M.D., Director—Postgraduate Division, School of Medicine, University of Southern California, 2025 Zonal Avenue, Los Angeles 33, California.

AMERICAN ROENTGEN RAY SOCIETY

SECTION ON INSTRUCTION

HAROLD O. PETERSON, M.D., *Director*

Titles and Abstracts of Courses Offered

Fifty-eighth Annual Meeting
Shoreham Hotel, Washington, D. C.
October 1–4, 1957

PRESIDENT-ELECT WENDELL G. SCOTT, with the approval of the Executive Council, has requested that the Section on Instruction of the American Roentgen Ray Society be continued for the Annual Meeting of 1957. The courses will all be of ninety minutes' duration and will be held between the hours of 3:00 P.M. and 4:30 P.M. on Tuesday, Wednesday and Thursday and from 1:30 P.M. to 3:00 P.M. on Friday. No other official activity has been scheduled for this time.

Fourteen rooms are available for the Instruction Courses and one course will be offered in each room each afternoon of the Annual Meeting. A total of 44 instructors will give fifty-four courses during the four days. Eight instructors in Diagnostic Roentgenology will present two courses and it is desirable, although not absolutely essential, that one should sign up for both courses. This is particularly true of the courses given by Doctors Hope, Etter, Dennis, Felson and Miller. In Therapeutic Radiology, the courses given by Doctors Fletcher and Bloedorn, as well as the two courses by Dr. Storaasli, should be considered as sequential courses and it would be advisable to sign up for all of the courses given by these instructors.

In summary, there are:

(1) Twenty Single Period Courses and Eight Two Period Courses in Diagnostic Roentgenology.

(2) Four Courses dealing with Nuclear Medicine.

(3) Nine Courses dealing with Therapeutic Radiology in general, including Roentgen-rays, Radium and Cobalt-60.

(4) Four Courses in Radiation Physics and Radiation Biology.

(5) One Course in Chemotherapy.

GENERAL INFORMATION

Location of Courses

The majority of the courses will be held in rooms which are numbered 1009, 1010, 1011, 1012, 1013, 1014, 1015, 1016, 1017 and 1018. These rooms are located very near the Registration Desk. Four other rooms will also be used on the same general floor level as the Registration Desk, designated as the North Room, the South Room, and the Green Room, near the West elevator, and the Tamerlane Room to the right of the Lower Lobby. Full information will be available at the desk of the Section on Instruction, which will be located in the General Registration Area on Sunday afternoon, Monday afternoon and from 8:30 A.M. until 4:30 P.M. Tuesday, Wednesday, Thursday and Friday.

How to Register and Obtain Tickets for the Instruction Courses

Admission to the Instruction Courses will be by ticket only.

Following the list of titles and abstracts is a general order sheet. First, second, and third choices for each period should be selected carefully and indicated on the order sheet. It is estimated that the number who can attend each course each day will range between 35 and 120 persons. If the directions given on the order sheet are followed explicitly, errors in completing reservations for the courses will be minimized.

It is possible for one to attend only four periods of instruction, so the condensed

schedule given on the last pages of this program should be consulted with care when registering and ordering tickets for the courses.

Reservations for the courses will be made in the order in which the order forms are received. Those who are not members of the American Roentgen Ray Society will be charged the nominal fee of $2.00 per period on instruction, or a maximum fee of $7.00 for four periods. Full-time graduate students in radiology will be admitted without fee, but they will be required to make application for the courses they wish to attend.

Previous to September 20, 1957, the order forms should be sent to the Director, Dr. Harold O. Peterson, 1995 West County Road "B," St. Paul 13, Minnesota. After September 20, 1957, the order forms should be sent directly to Dr. Harold O. Peterson, Shoreham Hotel, Washington, D. C.

To avoid the confusion always existing on the first days of the meeting, a set of tickets will be mailed to each person whose registration for courses reaches the office of the Director on or before September 20, 1957. The envelope containing the tickets will be labelled in a conspicuous manner. Registrants are urged to be on the lookout for their tickets, and to bring them to the meeting. The success of this plan depends entirely on the cooperation of the registrants.

In case the courses are not filled by the time of the meeting, tickets will be available at the Registration Desk of the Section on Instruction on Sunday, September 29, and thereafter during the meeting.

Holders of Tickets

Those who do not have the proper ticket for the course they wish to attend will not be permitted to enter the room. An Attendant will be at the door of each conference room to collect the tickets.

THE FACULTY

1. Fernando G. Bloedorn, M.D., Head, Division of Radiotherapy, Associate Professor of Radiology, University of Maryland, Baltimore, Maryland.
2. John Caffey, M.D., Professor of Radiology, College of Physicians and Surgeons, Columbia Presbyterian Medical Center, New York, New York.
3. Richard H. Chamberlain, M.D., Professor of Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.
4. Arthur E. Childe, M.D., Associate Professor of Radiology, University of Manitoba; Radiologist, Children's Hospital of Winnipeg; Associate Radiologist, Winnipeg General Hospital, Winnipeg, Manitoba, Canada.
5. John B. Coleman, M.D., Clinical Instructor in Radiology, University of Minnesota; Radiologist, Charles Miller Hospital, St. Paul, Minnesota.
6. John M. Dennis, M.D., Professor and Head, Department of Radiology, University of Maryland, Baltimore, Maryland.
7. Lewis E. Etter, M.D., Director, Department of Radiology, The Falk Clinic; Consultant, Western Psychiatric Institute and Clinic; Professor of Radiology, School of Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania.
8. Titus C. Evans, Ph.D., Head, Radiation Research Laboratory, College of Medicine, State University of Iowa, Iowa City, Iowa.
9. Benjamin Felson, M.D., Professor and Director, Department of Radiology, University of Cincinnati, Cincinnati, Ohio.
10. Gilbert H. Fletcher, M.D., Director of Radiation Therapy, M. D. Anderson Hospital and Tumor Institute, Houston, Texas.
11. Elizabeth F. Focht, B.A., Associate Attending Physicist, Memorial Center, New York, New York.
12. Hymer L. Friedell, M.D., Professor and Head of Radiology, Western Reserve University, Cleveland, Ohio.
13. Manuel Garcia, M.D., Director, Department of Therapeutic Radiology, Charity Hospital of Louisiana, New Orleans, Louisiana.
14. Alfred Gellhorn, M.D., Columbia University, College of Physicians and Surgeons, New York, New York.
15. Bentley Glass, Ph.D., Professor of Biology, Johns Hopkins University, Department of Biology, Baltimore, Maryland.

16. Torgny Greitz, M.D., Visiting Assistant Professor of Neuroradiology, Washington University School of Medicine, St. Louis, Missouri; formerly associated with Serafimer Hospital in Stockholm, Sweden.

17. Joseph Hanelin, M.D., Massachusetts General Hospital, Boston, Massachusetts.

18. Philip J. Hodes, M.D., Professor of Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.

19. Kenneth E. Hodge, M.D., F.R.C.P. (C), Hospital for Sick Children, Toronto, Ontario, Canada.

20. John W. Hope, M.D., Director, Department of Radiology, The Children's Hospital of Philadelphia; Assistant Professor of Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.

21. George Jacobson, M.D., Professor and Head of Radiology, University of Southern California, Los Angeles, California.

22. Peter James Kerley, M.D., F.R.C.P., F.F.R., Director, X-ray Department, Westminster Hospital; Consultant Radiologist, National Heart Hospital, London, England.

23. Richard G. Lester, M.D., Associate Professor of Radiology, University of Minnesota, Minneapolis, Minnesota.

24. R. Kenneth Loeffler, M.D., Director, Radiation Center, Madison, Wisconsin.

25. John S. Laughlin, Ph.D., Attending Physicist, Memorial Center, New York, New York.

26. Alexander R. Margulis, M.D., Assistant Professor of Radiology, University of Minnesota, Minneapolis, Minneota.

27. Alexander Stewart MacMillan, M.D., Director of Radiology Department, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts.

28. Jarrell E. Miller, M.D., Director of Radiology, Baylor University Hospital, Dallas, Texas.

29. Bernard J. O'Loughlin, M.D., Associate Professor of Radiology, Head, Diagnostic Division, University of California Medical Center, Los Angeles, California.

30. Francis F. Ruzicka, Jr., M.D., Director of Radiology, St. Vincent's Hospital of the City of New York; Clinical Professor of Radiology, New York University College of Medicine, New York, New York.

31. Richard Schatzki, M.D., Chief, Department of Radiology, Mount Auburn Hospital, Cambridge, Massachusetts; Clinical Associate in Radiology, Harvard Medical School, Boston, Massachusetts.

32. Robert Shapiro, M.D., Director, Department of Radiology, The Hospital of St. Raphael, New Haven, Connecticut.

33. Glen E. Sheline, M.D., Department of Radiology, University of California Medical Center, San Francisco, California.

34. Norman Simon, M.D., Assistant Attending Radiotherapist, Mt. Sinai Hospital, New York, New York.

35. David Waldron Smithers, M.D., F.R.C.P., D.M.R., F.F.R., Director, Radiotherapy Department, The Royal Marsden Hospital, London, England.

36. Herbert M. Stauffer, M.D., Professor and Director, Diagnostic Division, Department of Radiology, Temple University Hospital, Philadelphia, Pennsylvania.

37. John P. Storaasli, M.D., Western Reserve University, Cleveland, Ohio.

38. William J. Tuddenham, M.D., Assistant Professor of Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.

39. Eugene F. Van Epps, M.D., Professor and Director, Department of Radiology, University of Iowa, Iowa City, Iowa.

40. W. Walter Wasson, M.D., Denver, Colorado.

41. Harry M. Weber, M.D., Director, Section on Roentgenology, Mayo Clinic, Rochester, Minnesota.

42. Robert E. Wise, M.D., Department of Radiology, The Lahey Clinic, and New England Baptist Hosptial, Boston, Massachusetts.

43. Martin H. Wittenborg, M.D., Children's Hospital; Assistant Clinical Professor, Harvard Medical School, Boston, Massachusetts.

44. Henry J. Woloshin, M.D., Assistant Professor of Radiology, Temple University, Philadelphia, Pennsylvania.

DESCRIPTION OF COURSES

COURSE 100

Tuesday

PETER JAMES KERLEY, M.D., F.R.C.P., F.F.R.
London, England

System of Cardiovascular Roentgenology in the National Heart Hospital, London

1. Techniques in the National Heart Hospital.
2. Direct diagnosis of congenital cardiovascular lesions.
3. Diagnosis of the nonrheumatic cardiac myopathies.
4. Diagnosis of dissecting and nonspecific aneurysms of the great vessels.

COURSE 101 and COURSE 201

Tuesday and Wednesday

JOSEPH HANELIN, M.D.
Boston, Massachusetts

Cerebral Calcifications and Plain Film Findings of Brain Tumors

Course 101. Cerebral Calcifications. Gross calcium deposition may occur in a large group of intracranial lesions. The type and distribution of this calcification at times is quite distinctive, and at the very least should always evoke a carefully considered differential diagnosis. Conditions under discussion will include those of degenerative, vascular, infectious, traumatic and neoplastic etiology. Clinical correlation will be emphasized.

Course 201. Plain Film Findings of Brain Tumors. Despite the free use of pneumography and angiography in the study of intracranial lesions, it is important to recognize and re-emphasize the value of the comparatively simple and innocuous plain film skull examination. This session will be devoted to a review of the pertinent changes in the calvarium and base of the skull as well as to displacement of physiologic calcification in the diagnosis of space-occupying masses. Certain normal variations which may simulate such abnormality will also be discussed.

Those attending either of these courses are urged to bring along any interesting or puzzling roentgenograms pertaining to the subject which will add interest to the discussion period at the close of each lecture.

COURSE 102 and COURSE 202

Tuesday and Wednesday

JOHN W. HOPE, M.D.
Philadelphia, Pennsylvania

Techniques of Pediatric Radiology and Pediatric Radiologic Problems

Most departments of radiology are geared both physically and mentally to handle adults. That infants and children and the diseases and abnormalities with which they may be afflicted differ greatly from grown-ups is not generally appreciated. All too often a patient is referred to The Children's Hospital of Philadelphia because a roentgenogram has been said to show some serious abnormality. The parents of these children are terrified and often almost hysterical. With more careful technique and interpretation these serious abnormalities often resolve into perfectly normal findings. This course is designed to show some of these pitfalls, and to suggest how to avoid them.

Course 102 will deal with the chest, the neck, intravenous urography, and the skull.

Course 202 will deal with the abdomen and the gastrointestinal tract.

COURSE 103 and COURSE 203

Tuesday and Wednesday

ALEXANDER STEWART MACMILLAN, M.D.
Boston, Massachusetts

Sinuses and Mastoids, Lateral Neck and Larynx

Course 103. Sinuses.
Course 203. Mastoids, Lateral Neck and Larynx.

COURSE 104

Tuesday

ROBERT SHAPIRO, M.D.
New Haven, Connecticut

Metabolic Bone Diseases

Traditionally, much emphasis has been placed upon the role of the skeleton as a supporting structure. Considerably less attention has been focused on the homeostatic role of the skeleton in maintaining the composition of the body fluids. Because of its active participation in many important metabolic processes, the skeleton is subject to change in a number of systemic diseases. This course will attempt to present a logical classification of metabolic bone diseases and

correlate the roentgen findings with the physiologic and chemical abnormalities.

COURSE 105

Tuesday

W. WALTER WASSON, M.D.
Denver, Colorado

The Physio-Dynamics of the Lesser Circulation with Their Practical Application as Found in Daily Practice and as of Interest to the Radiologist, the Clinician

I. A brief historical sketch of the researches which have made this presentation possible.

II. The anatomy and the physiology of the lungs and the physio-dynamics which concern the lesser circulation.

III. Discussion of cases illustrating the influences of the physio-dynamics of the lesser circulation as follows:

A. Cor pulmonale in emphysema and lung fibrosis
B. Sudden death from lung embolus
C. The diaphragmatic syndrome and obesity
D. The closure of the ductus arteriosus
E. Hypoventilation and atelectasis
F. Mitral disease and lung congestion
G. Kyphosis and heart failure

COURSE 106

Tuesday

ARTHUR E. CHILDE, M.D.
Winnipeg, Manitoba, Canada

Intussusception in Infants and Children

The method of choice in the treatment of intussusception is still a controversial matter. Operative reduction was first successfully performed eighty-three years ago and since then many authors have advocated surgical reduction in all cases. Others have used conservative methods of reduction, first air or water enemas, and more recently, barium enemas.

In this course the entire problem will be discussed and the experience over an eight year period at a children's hospital will be outlined. During this time almost all cases suspected to have intussusception have received a barium enema for diagnostic purposes, and if such a lesion has been found attempts have been made to reduce it by hydrostatic pressure.

COURSE 107

Tuesday

WILLIAM J. TUDDENHAM, M.D.
Philadelphia, Pennsylvania

New Concepts in Radiographic and Interpretative Techniques

The inadequacy of "density," "resolution," "contrast" and "unsharpness" as criteria for evaluating the "quality" of diagnostic roentgenograms will be discussed, and new criteria of "film quality" will be presented.

The significance of the various physical factors affecting the "quality" of roentgenograms will be considered, and the indications for so-called "high-kilovoltage" and "supervoltage" radiography as well as for the use of the "micro-focus" roentgen-ray tube will be specifically noted.

Recent observations concerning view-box and surrounding illumination, the control of glare, the choice of reading lenses—practical aids for the radiologist in perceiving critical "threshold" images—will be presented.

Pertinent experimental findings will be cited, but the practical clinical application of these concepts will be emphasized through the presentation of illustrative diagnostic roentgenograms.

COURSE 108

Tuesday

GEORGE JACOBSON, M.D.
Los Angeles, California

Roentgen Diagnosis of Injuries of the Atlanto-Axial Joint

COURSE 200

Wednesday

HARRY M. WEBER, M.D.
Rochester, Minnesota

One Person's Views on the Intestinal Polypoid Lesion

The period will be devoted to a general discussion of the various roentgenologic maneuvers currently in vogue to demonstrate the excrescences on the mucous membrane of the colon commonly known as intestinal polyps. The significance of such lesions will be reviewed in some detail, and a commentary will be made on certain therapeutic implications. No argument for any one particular roentgenologic procedure will be offered, since all of them have the same ultimate aim, and all seem to have their own peculiar advantages and limitations. These will receive detailed attention.

COURSE 204

Wednesday

RICHARD SCHATZKI, M.D.
Cambridge, Massachusetts

The Use of Gravity Maneuvers in the Examination of the Stomach

The use of gravity in the study of the stomach was forced upon the radiologist for the examination of massively bleeding patients. The inadvisability of palpation, far from being a handicap, proved to be a blessing in disguise necessitating various maneuvers to examine every part of the stomach successfully.

These procedures have been found helpful for practical use in the examination of all stomachs. They have proved particularly valuable in the study of the upper part of the stomach.

Systematic examination of the stomach with such gravity maneuvers will be described. Its practical application in the differential diagnosis of ulcer, cancer and other lesions of the stomach will be discussed.

COURSE 205

Wednesday

HERBERT M. STAUFFER, M.D.
Philadelphia, Pennsylvania

Experimental Cinefluorographic Heart Studies

Motion picture films will be shown illustrating the use of cinefluorography in experimental studies of the structure and function of the dog heart. A standard 16 mm. motion picture camera has been used in connection with image intensifiers.

The following experimental studies will be covered: (1) Intracardiac carbon dioxide for visualization of the right and left heart. (2) Variations of mitral valve competence in normal dogs. (3) Visualization of effects of experimental supravalvular pulmonic stenosis. (4) Intracardiac carbon dioxide demonstration of pericardial fluid.

COURSE 206

Wednesday

JOHN B. COLEMAN, M.D.
St. Paul, Minnesota

Roentgen Anatomic Studies of the Esophagus; A Method for General Use; and Application to the Diagnosis and Exclusion of Hiatus Hernia

Routine detailed study of the lower esophagus is feasible in the upper gastrointestinal tract examination. The most difficult part of the method lies in obtaining good spot roentgenograms, since the normal or pathologic features usually appear only briefly, and in securing representative serial roentgenograms of the esophageal dynamics.

Anatomically, the lower segment of the esophagus above the diaphragm, commonly referred to as the phrenic ampulla, seems a structure not wholly esophageal or gastric. It is bounded below by a sphincter of greater or lesser competency which is likely formed by the crura. The ampulla is bounded above by a circular constrictor beyond which the peristaltic wave of the esophagus probably does not travel. This constrictor is more frequently seen under proper conditions than the sphincter at the diaphragm. Between these anatomic landmarks a ring may appear with a frequency of 10 to 22 per cent. In various forms it is known as the ampullary ring or Schatzki-Gary ring. The latter is probably the symptomatic form. The ring may be deep, shallow, complete, partial and, perhaps, multiple. When well defined the ring often marks a functional division between upper and lower halves of the ampulla. These halves, or segments, may collapse at different rates and produce thereby an appearance of small hiatus hernia.

The nature of the mucosal folds resembling gastric folds above the diaphragm is conjectured. It seems possible that the anatomy of the lower end of the supradiaphragmatic esophagus is altered in the later decades of life. The occurrence of superimposed adult-type pathology and symptoms is postulated. The possibility that the esophageal constrictor at the upper end of the ampulla is converted to an important functional shut-off is raised. The need for accurate correlative studies is emphasized.

The various forms of the normal esophagus which imitate hiatus hernia will be demonstrated and discussed. The fluoroscopic and spot-roentgenographic techniques necessary will be described. Familiarity with the varied features of the ampulla is essential and for that reason care will be taken to show in the discussion the development of "false hernias" from normal esophageal structures on films. The confirmation of small hernias which might otherwise be equivocal will be demonstrated.

COURSE 207
and
COURSE 307

Wednesday and Thursday

LEWIS E. ETTER, M.D.
Pittsburgh, Pennsylvania

Roentgen Anatomy of the Skull

Course 207. Description of standard techniques and positions with illustrative slides for roentgen examination of the skull and paranasal sinuses. Comparison of advantages of one projection over another with respect to visibility of particular structures. General considerations with reference to the cerebral and facial components of the cranium and various

skull types. Analysis of individual bone components in producing the various lines and shadows seen in plain roentgenograms of the skull. This analysis will be applied to a study of the sphenoid, frontal, ethmoid, parietal and occipital bones in various projections.

Course 307. Detailed roentgen anatomic analysis of the paranasal sinuses and mastoids with description of roentgenographic techniques employed. Study of the roentgen features of the middle cranial fossa and their relation to the sphenoid sinuses. Consideration of the orbits and optic foramina with special attention to the frontal, ethmoid and sphenoid bone components in various projections as well as the features contributed by the zygomatic bones. In conclusion, the maxillofacial components of the cranium will be examined, including the lacrimal canals and soft tissue delineation.

COURSE 208

Wednesday

KENNETH E. HODGE, M.D., F.R.C.P. (C)
Toronto, Ontario, Canada

The Radiologic Diagnosis of Placenta Previa

A modification of Reid's gravitational method of placental localization and its practical applications are described. Other methods that have been employed are also briefly discussed.

A series of 200 suspected cases of placenta previa examined by the gravitational method is reviewed. An accuracy of 95 per cent in the diagnosis and in the exclusion of placenta previa has been achieved.

COURSE 300

Thursday

JOHN CAFFEY. M.D.
New York, New York

Normal Variants in the Growing Skeleton of Diagnostic Significance

1. Estimation of bone age
2. Prenatal ossification and estimation of prematurity
3. Local anatomic variants
   a. Hand
   b. Bones of the arm
   c. Bones of the legs
      (1) Benign cortical defects
   d. Bones of the pelvis
      (1) Acetabular angles in congenital dislocation of the hip
      (2) Swellings of the ischiopubic synchondrosis
      (3) Irregular mineralization of the neonatal pubic bones
   e. Cranium
      (1) Neonatal sutures
      (2) Interparietal bones
      (3) Accessory ossicles of occipital bone
      (4) Marginal irregularities of parietal bones

COURSE 301 and COURSE 401

Thursday and Friday

JOHN M. DENNIS, M.D.
Baltimore, Maryland

Problems in Urinary Tract Diagnosis

Course 301. Techniques employed in the diagnosis in urinary tract lesions will be briefly discussed. While aortography, retroperitoneal air studies, nephrotomography and retrograde urography will be covered, emphasis will be placed upon obtaining maximum information from intravenous urographic studies by employing a few additional simple procedures and the information obtained from them will be shown.

Following a discussion of techniques, many diagnostic problems of the urinary tract will be presented. Emphasis will be placed upon the diagnosis of these lesions by ordinary urographic procedures.

Course 401. A continuation of the discussion of diagnostic problems of the urinary tract will be presented.

COURSE 302 and COURSE 402

Thursday and Friday

BENJAMIN FELSON, M.D.
Cincinnati, Ohio

Some Fundamentals in Chest Roentgen Interpretation

Chest roentgen interpretation should not merely represent an attempt to correlate particular shadows with specific diseases, but should be approached with an understanding of the basic principles of anatomy, physiology, pathology, and radiology involved. Some of these principles will be considered and their practical application illustrated.

Among the subjects to be discussed are:

Course 302. (1) A method of fluoroscopy; (2) the "silhouette" sign; and (3) the air bronchogram.

Course 402. (1) Anatomic variations in the pulmonary fissures; (2) lobar collapse; (3) lobar enlargement; and (4) the disrupted fissure.

COURSE 303

Thursday

HENRY J. WOLOSHIN, M.D.
Philadelphia, Pennsylvania

Ulcerative Colitis

The roentgen examination of the colon plays an important role in the over-all picture of ulcerative colitis. Although the diagnosis may be established before the patient is referred for roentgen study, the barium enema examination allows one to estimate the extent and severity of involvement of the colon. The earliest roentgen signs of ulcerative colitis will be demonstrated. There will be a discussion of pseudopolyposis, ileocecal involvement, segmental involvement, and the occurrence of carcinoma. The chronic course of the disease in two cases will be presented.

COURSE 304

Thursday

BERNARD J. O'LOUGHLIN, M.D.
Los Angeles, California

Selective Angiocardiography

The relative merits of selective versus venous and retrograde methods of visualizing the heart and great vessels will be discussed, giving the results of our experiences, with some suggestions on how to avoid errors into which we have fallen and best methods of achieving good diagnostic studies. The problems of contrast materials and radiation hazards will be discussed.

A résumé of the diagnostic features of the various lesions which we have encountered in illustrative cases will be presented. Time will be available for discussion and questions.

COURSE 305

Thursday

ROBERT E. WISE, M.D.
Boston, Massachusetts

Intravenous Cholangiography

This course will review experiences with over 1,000 intravenous cholangiographies, the majority of which have been carried out on patients who had previously had cholecystectomy. Technique and reactions will be discussed. The numerous pitfalls encountered in the procedure will be pointed out. Diagnostic aspects of the procedure in the precholecystectomy patient related to both the gallbladder and the biliary ducts will be included. Since the procedure has been found to be most useful in the postcholecystectomy patient, the major portion of the time will be utilized in a discussion of the findings in this group of patients. Data related to the size and drainage of the common bile duct, obstruction (fibrosis of the sphincter of Oddi, calculi, tumors, and strictures) will be analyzed and correlated with operative findings in over 15 per cent of the entire group. Particular emphasis will be placed upon the estimation of drainage of the ducts as it is related to the above conditions.

The procedure has been of considerable value in leading to definitive surgical treatment and subsequent relief in cases of partial obstructions of the biliary tract. Perhaps of even greater importance, however, has been its value in eliminating needless exploratory operations on the biliary tract.

COURSE 306
and
COURSE 406

Thursday and Friday

JARRELL E. MILLER, M.D.
Dallas, Texas

Roentgen Examination of the Stomach and Small Bowel

The roentgen examination of the stomach and small bowel is important. Not all radiologists are able to maintain adequate skill in interpretation of special studies such as cerebral angiography, pneumography, angiocardiography and other similar procedures when they have little or no opportunity to see them. The upper gastrointestinal tract study, however, is one of the most frequently performed examinations where the radiologist plays a dominant role. It is necessary, therefore, that he acquire and maintain a superior skill in performing these examinations as well as interpreting the results.

Course 306 will deal principally with techniques of the upper gastrointestinal tract and small bowel examination. The minimum film requirement for an adequate upper gastrointestinal tract study will be demonstrated. Techniques for combination procedures which can be done without compromising any of the examinations will also be shown. Pitfalls and errors in interpretation will be discussed along with methods of avoiding them.

Course 406 will comprise the demonstration of the various lesions which can be diagnosed by means of this examination plus the unusual lesions which one encounters in a busy practice. If the group desires, the last half hour of the second period will be used for a quiz session.

COURSE 308
and
COURSE 408

Thursday and Friday

TORGNY GREITZ, M.D.
St. Louis, Missouri
(Stockholm, Sweden)

Pneumography—Technique and Anatomy
and
Pneumography in Expanding Lesions of the Brain

Course 308. Pneumography—Technique and Anatomy. The indications for encephalography and ventriculography, as carried out at the Serafimer Hospital in Stockholm, will be briefly described. This course also will deal with the normal anatomy and the variations of the ventricular system and the subarachnoid cisterns.

Course 408. Pneumography in Expanding Lesions of the Brain. Localizations of tumors by general and local deformity of the ventricles and the subarachnoid cisterns will be discussed with special emphasis placed upon tumors of the temporal lobe and the posterior fossa.

COURSE 400

Friday

PHILIP J. HODES, M.D.
Philadelphia, Pennsylvania

Recent Advances in Body Section Radiography

There are many factors which bear upon effective and informative body section radiography. There are fundamentals without which the modality can prove to be a "hit or miss" proposition. In addition to the ordinary techniques, horizontal and erect positions, one is now aided immeasurably by the "book cassette" which makes it possible to save a great deal of time and radiation exposure without losing any information in the finished radiograph.

The fundamentals will be reviewed and considerable time will be spent in discussing the "book cassette." Demonstration of clinical applications of the modality will complete the session.

COURSE 403

Friday

FRANCIS F. RUZICKA, JR., M.D.
New York, New York

Portal Venography—Techniques, Interpretation and Clinical Application

Portal venography is a roentgen method which in a relatively short time has become useful in the study of abnormalities of the portal venous system and has earned an accepted position in the medical armamentarium of diagnostic procedures. It has practical application in the clinical problems of portal venous obstruction of both intrahepatic and extrahepatic types.

Cirrhosis (Laennec's cirrhosis in particular) is the most common cause of portal obstruction of the intrahepatic type. Portal hypertension with serious and frequently fatal gastrointestinal bleeding is one of the sequelae. The most effective means of therapy so far devised for the treatment of bleeding gastroesophageal varices is portacaval surgery. The portal venogram is of inestimable value to the surgeon in revealing immediate and detailed information concerning the anatomy and physiology of the portal venous system. Much operating time can thereby be saved by avoiding painstaking and difficult exploration, and indeed the venogram is of great aid in planning the operative procedure.

In the extrahepatic type of portal obstruction, the same is true. The site of venous block is readily demonstrated and the region of venous anatomy is defined. Location of a mass may be determined or confirmed. These and other practical applications of portal venography will be considered in this course.

Special attention will be paid to the technique of splenic portography performed percutaneously in the radiologic department, as well as to the portal portography performed at laparotomy in the operating room.

Normal and pathologic anatomy and physiology of the portal venous system will be reviewed to form a background for interpretation of the portal venogram. Features to be emphasized will include the site of venous block, collateral vein development, the streaming phenomenon, and other factors which influence roentgen visualization of the portal venous system.

COURSE 404

Friday

RICHARD G. LESTER, M.D.
Minneapolis, Minnestoa

Roentgen Diagnosis of Cardiac Lesions in Infants and Children

Since the advent of corrective surgical procedures for many of the cardiac lesions of childhood, the necessity for accurate diagnosis in these patients has become most important. In this course, the place of roentgenology in the clinical examination of patients with heart disease in the pediatric age range will be outlined. Interpretation in terms of pathologic anatomy and physiology will be stressed.

Correlation of the roentgen findings with the other clinical procedures and examinations will be presented, and the team approach to diagnosis of cardiac lesions emphasized. The use of conventional roentgen techniques, their value and limitations will be discussed at length. Special procedures, such as angiocardiography and retrograde aortography will be

used for confirmation and further discussion, but this course will be oriented to the methods available to all radiologists.

COURSE 405

Friday

ALEXANDER R. MARGULIS, M.D.
Minneapolis, Minnesota

Arteriography of Extremities

The indications and value of arteriography of extremities will be discussed. Technique and contrast medial will be considered in detail. The normal anatomy will be presented and features of various peripheral vascular diseases will be outlined. The importance of studying the small vessels as well as the large arteries will be stressed. Occlusive disease, arteriovenous communications, bone and soft tissue tumors will also be discussed. It will be emphasized that peripheral arteriography, due to progress in technique, has become a relatively harmless procedure which can be done on an out-patient basis.

COURSE 407

Friday

EUGENE F. VAN EPPS, M.D.
Iowa City, Iowa

Diagnosis of Pneumonias in Infancy and Early Childhood

This course will present the correlation of the roentgenologic appearance of acute pneumonias, especially in infants, with the pathologic findings. An attempt will be made to clarify the errors made by radiologists in calling a chest film with severe pneumonitis normal, because it looks so clear, and the error of calling a healthy chest abnormal.

COURSE 120
COURSE 220
and
COURSE 320

Tuesday, Wednesday and Thursday

GILBERT H. FLETCHER, M.D.
Houston, Texas
and
FERNANDO G. BLOEDORN, M.D.
Baltimore, Maryland

Radiotherapeutic Management of Oral Cavity and Oral Pharynx Cancers

Course 120. A. Biologic and Physical Factors: Histology of tumors, condition of tumor bed, concept of lethal dose, and other factors to consider in prescribing treatment (conventional and supervoltage beam, radium).

B. Principles of Beam Therapy Techniques (Physical Factors): (1) Simple stationary portals; (2) multiple beam techniques with and without cases; (3) wedge filter beams; (4) rotation; and (5) intraoral therapy.

C. Radium Implants: (1) Indications; (2) different types of gamma rays and emitting isotopes; (3) distribution of radium in needles; (4) technique and implantation; (5) dosimetry; and (6) dose-time-volume relationship.

Mimeographed material on the following will be available for the audience: (1) Radium implant arrangements; (2) common patterns; (3) indication for radium; (4) patterns of special isotopes.

Courses 220 and 320 will consist of a discussion of selected cases covering the most commonly encountered situations and illustrating the application of general principles of radium therapy and external irradiation. The clinical factors determining indications and techniques are: (1) Histology; (2) site of the disease; (3) exact location for a particular site; (4) extent of the disease; (5) extension to surrounding structures; and (6) behavior of lymphatic spread.

Course 220 will cover the following sites of the oral cavity: (1) Anterior two-thirds of the tongue; (2) floor of the mouth; (3) buccal mucosa; and (4) gums.

Course 320 will cover the following sites of the oral pharynx: (1) Retromolar trigone; (2) faucial pillars; (3) glossopalatine sulcus; (4) base of tongue; (5) tonsils; (6) soft palate; and (7) pharyngeal walls.

COURSE 121

Tuesday

RICHARD H. CHAMBERLAIN, M.D.
Philadelphia, Pennsylvania

The Isotope Laboratory and Its Management

Radiologists are well equipped by their background to be central figures in the clinical uses of the radioactive isotopes, but this position requires much knowledge and experience in addition to what is needed for conventional diagnostic and therapeutic radiologic practice. The "What?", "How?", and "Where?" of the preparation for the practical use of radioactive isotopes will be discussed. The problems connected with procurement and arrangement of laboratory facilities and equipment, the training of technical personnel, and organization of the general program will also receive attention. General uses of radioisotopes in diagnosis, therapy and teaching will be discussed as time permits.

COURSE 122

Tuesday

NORMAN SIMON, M.D.
New York, New York

Cobalt 60 Teletherapy: Treatment Techniques with a Hectocurie Unit

Cobalt 60 hectocurie teletherapy promises to replace 250 kv. roentgen-ray therapy as "conven-

tional" radiotherapy. Changes in technique of treatment are required because of the characteristics of cobalt 60 radiation, i.e. sparing of skin, relatively low bone absorption and decreased side scatter. The effect of these characteristics on treatment plans for cancers of the head and neck, breast, lung, esophagus, uterus and rectum will be discussed.

The limitations of hectocurie cobalt 60 teletherapy will also be presented.

COURSE 123

Tuesday

BENTLEY GLASS, PH.D.
Baltimore, Maryland

The Genetic Effects of Radiation

According to overwhelming evidence, genetic information is transmitted from one generation to the next through the chemical medium of deoxyribose nucleic acid (DNA), which forms a major part of the chromosomes. Permanent genetic alterations, that is to say, mutations, consist of modifications of the chromosomes and specifically of the sequences of purine and pyrimidine bases (adenine, guanine, thymine, cytosine) in the DNA. Microscopically visible breakage and rearrangement of chromosomes is produced by all sorts of ionizing radiations, and may consist of translocations of chromosome segments, inversions, duplications, or deficiencies. Submicroscopic mutations must be basically of a similar nature; that is, shifts in the arrangement of base pairs, inversions of segments, insertions, deletions, and substitutions. Because of the capacity of DNA to replicate itself, such changes, once produced, replicate themselves and are inherited. The gross chromosomal aberrations increase as the square or some higher power of the ionization per unit volume produced by radiations such as cosmic radiation, gamma rays and roentgen rays, and by ionizing particles such as beta particles, alpha particles, and neutrons. This is presumable because separate quanta or particles are required for separate breaks; but neutrons and alpha particles have a higher efficiency because one particle may have a fair probability of breaking two adjacent chromosome strands. Submicroscopic mutations seem always, on the other hand, to increase linearly with the radiation dose. Down to the lowest measurable doses there is no sign of a threshold; that is, a single high-energy quantum appears capable of producing a mutation and the probability is equal for all which traverse the target. The effectiveness of chemical mutagens in producing mutations, and the reduction of mutation frequency for a given dose when roentgen rays are administered in the absence of oxygen and the converse increase when they are administered in pure oxygen instead of air, together indicate that the high-energy quantum need not score a direct hit on the DNA to produce a mutation, but the indirect pathways of effect may also result in mutations.

Mutations, the vast majority of which are harmful to some degree but largely recessive in effect, also occur spontaneously. It is a serious unsolved problem to determine what fraction of the spontaneous mutation is attributable to the background radiation.

The amount of the background radiation in the United States and some areas of the earth (Travancore, Brazil) will be discussed and the dose of radiation that would double the spontaneous mutation rate will be considered.

A doubling dose would seriously increase the genetic defectiveness of any population exposed to it, and the upper limit of radiation to which the entire population should be exposed ought to be kept well below a doubling dose. This was the basis of the recommendation by the National Academy of Science's Genetics Committee of an upper limit of 10 r gonadal dose over the thirty year average reproductive lifetime.

COURSE 124

Tuesday

R. KENNETH LOEFFLER, M.D.
Madison, Wisconsin

Calculation of Radium Dosage

Principles of radium dosage are developed from an analysis of the simplest geometric forms—the point, line, plane, ring and sphere. The radiation distribution about each is unique and simple, but each acts effectively as a point source contributor to a moderately distant point. From these considerations, the use of the standard dosage tables is developed, with roentgenograms of implants and discussion of the clinical features.

COURSE 221
and
COURSE 321

Wednesday and Thursday

DAVID WALDRON SMITHERS, M.D.
F.R.C.P., D.M.R., F.F.R.
London, England

Some Etiologic and Therapeutic Aspects of Carcinoma of the Esophagus
and
The Treatment of Hodgkin's Disease

Course 221. Some Etiologic and Therapeutic Aspects of Carcinoma of the Esophagus. The association of such conditions as achalasia and hiatus hernia and reflux esophagitis with esophageal cancer will be discussed. The role of radiation therapy and of surgery in the treatment of carcinoma of the esophagus arising at different sites will be assessed and a review of the results obtained by both methods all over the world presented. A view of the future possibilities of advances in the treatment of cancer of the esophagus will be put forward.

Course 321. The Treatment of Hodgkin's Disease. An assessment will be attempted of some of the factors influencing the prognosis in Hodgkin's disease. Survival will be related to histology, site of first detected lesion, dissemination, multifocal origin, general symptoms, duration of symptoms, sex, pregnancy, age, stage, and method of treatment. The use of radiation therapy and chemotherapy in modifying the course of the disease and in relieving or prolonging suffering will be discussed.

COURSE 222

Wednesday

ALFRED GELLHORN, M.D.
New York, New York

Chemotherapy in Disseminated Neoplastic Disease (Including Lymphomas)

The material to be covered includes a discussion of the indications, contraindications, and toxicity of drugs in the treatment of disseminated neoplastic disease. Emphasis will be placed on the use of hormones, antimetabolites and alkylating drugs. The relationship of chemotherapy and radiation therapy in the management of human cancer and lymphomas will be stressed.

COURSE 223

Wednesday

ELIZABETH F. FOCHT, B.A.
New York, New York

Dosimetry No. 1—Internally Administered Radioisotopes

A summary of the methods and data necessary to obtain the dose roentgens, equivalent roentgens or rads from any isotope will be analyzed as follows:

A. Isotope source. Gamma and/or beta. Millicuries, energy, effective half-life.
B. Distribution. Discrete sources. Localized or uniformly distributed solutions.
C. Calculations. I_Y and D_B. Available tables, charts and references.
D. Typical problems for therapy. Analysis of tracer amounts allowable within protection limits.

COURSE 224

Wednesday

HYMER L. FRIEDELL, M.D.
Cleveland, Ohio

Visualization of Internal Structures by Special Scanning Techniques

The use of special scanning techniques for visualizing the liver and certain blood masses will be discussed. Liver disease of various kinds will be demonstrated, including metastatic disease and other space-occupying lesions.

The technique for demonstrating blood masses and visualizing unusual cardiac conditions, as well as aneurysms, will also be shown.

The description of the technique will include discussion of a regular printing method, as well as an oscilloscopic method.

COURSE 322
and
COURSE 422

Thursday and Friday

JOHN P. STORAASLI, M.D.
Cleveland, Ohio

Nuclear Medicine: Therapeutic Applications

A survey will be made of the more important therapeutic applications of radioisotopes. To be included will be a discussion of the rationale of therapy and a comparison of other methods of treatment. The isotopes to be covered in detail will be radioactive phosphorus in the treatment of polycythemia, leukemia and metastatic breast carcinoma in bone; radioactive iodine for diseases of the thyroid gland and euthyroid cardiacs; radioactive colloidal gold for malignant serous effusions and for interstitial administrations; radioactive strontium for beta-ray therapy of ocular lesions; and radioactive cobalt in certain types of applicators.

COURSE 323

Thursday

JOHN S. LAUGHLIN, Ph.D.
New York, New York

Dosimetry No. 2—External Radiation Therapy

The concepts and use of air dose, skin dose, exposure dose and absorbed dose will be described for both conventional roentgen-ray therapy and high energy roentgen-ray and electron therapy. The dose distributions produced by various sources and treatment planning procedures will be described. Elements of radiation protection will also be presented.

COURSE 324

Thursday

GLEN E. SHELINE, M.D.
San Francisco, California

Radioiodine in Thyroid Disease

This discussion will include both diagnostic and therapeutic uses of radioiodine in thyroid disease; however, more emphasis will probably be placed on the therapeutic aspects. The thyroid uptake of I^{131} in various thyroid diseases will be considered and ex-

amples of scanning for nodules and aberrant thyroid tissue will be shown. A discussion of the place of radioiodine in the treatment of thyrotoxicosis and thyroid carcinoma will be given. The discussion will include indications, limitations and results of radioiodine therapy for these diseases.

COURSE 420

Friday

MANUEL GARCIA, M.D.
New Orleans, Louisiana

The Treatment of Carcinoma of the Cervix

This course will present an explanation of the techniques of roentgen-ray and radium therapy used at the Charity Hospital of New Orleans, Louisiana, in the management of cases of carcinoma of the cervix. The methods will be described. Dosimetry will be considered in relation to biologic effectiveness, and the variations in the radiation program required by the presence of several complications will be discussed. Recommendations on treatment will be supported by an analysis of the end results.

COURSE 421

Friday

MARTIN H. WITTENBORG, M.D.
Boston, Massachusetts

Radiotherapeutic Aspects of Tumors in Infants and Children

The types of tumors encountered in children will be reviewed. Emphasis will be placed on the malignant tumors, their growth characteristics, their so-called radiosensitivity, and the radiation tolerance of the child and his growing parts. The problems will be discussed primarily on an empirical clinical basis.

COURSE 423

Friday

TITUS C. EVANS, PH.D.
Iowa City, Iowa

Radiation Biology

Current concepts and recent literature concerning biologic effects of ionizing radiation will be discussed. Topics such as mechanism of action, effects of protraction, fractionation, linear energy transfer, modifying agents, etc., will be examined with a view to applying them as much as possible to radiation therapy and radiation protection problems.

CONDENSED SCHEDULE OF COURSES

TUESDAY **3:00 to 4:30 P.M.** **October 1, 1957**

Diagnostic Roentgenology		Nuclear Medicine, Therapeutic Radiology, Miscellaneous	
100—Kerley	System of Cardiovascular Roentgenology in the National Heart Hospital, London	**120—Fletcher and Bloedorn**	Radiotherapeutic Management of Oral Cavity and Oral Pharynx Cancers
101—Hanelin	Cerebral Calcifications		
102—Hope	Techniques of Pediatric Radiology	**121—Chamberlain**	The Isotope Laboratory and Its Management
103—MacMillan	Sinuses		
104—Shapiro	Metabolic Bone Disease		
105—Wasson	The Physio-Dynamics of the Lesser Circulation with Their Practical Application as Found in Daily Practice and As of Interest to the Radiologist, the Clinician	**122—Simon**	Cobalt 60 Teletherapy: Treatment Techniques with a Hectocurie Unit
106—Childe	Intussusception in Infants and Children	**123—Glass**	The Genetic Effects of Radiation
107—Tuddenham	New Concepts in Radiographic and Interpretative Techniques		
108—Jacobson	Roentgen Diagnosis of Injuries of the Atlanto-Axial Joint	**124—Loeffler**	Calculation of Radium Dosage

CONDENSED SCHEDULE OF COURSES

WEDNESDAY **3:00 to 4:30 P.M.** **October 2, 1957**

Diagnostic Roentgenology		Nuclear Medicine, Therapeutic Radiology, Miscellaneous	
200—Weber	One Person's Views on the Intestinal Polypoid Lesion	**220—Fletcher and Bloedorn**	Radiotherapeutic Management of Oral Cavity and Oral Pharynx Cancers
201—Hanelin	Plain Film Findings of Brain Tumors		
202—Hope	Pediatric Radiologic Problems	**221—Smithers**	Some Etiologic and Therapeutic Aspects of Carcinoma of the Esophagus
203—MacMillan	Mastoids, Lateral Neck and Larynx		
204—Schatzki	The Use of Gravity Maneuvers in the Examination of the Stomach	**222—Gellhorn**	Chemotherapy in Disseminated Neoplastic Disease (Including Lymphomas)
205—Stauffer	Experimental Cinefluorographic Heart Studies		
206—Coleman	Roentgen Anatomic Studies of the Esophagus; A Method for General Use; and Application to the Diagnosis and Exclusion of Hiatus Hernia	**223—Focht**	Dosimetry No. 1—Internally Administered Radioisotopes
207—Etter	Roentgen Anatomy of the Skull	**224—Friedell**	Visualization of Internal Structures by Special Scanning Techniques
208—Hodge	The Radiologic Diagnosis of Placenta Previa		

CONDENSED SCHEDULE OF COURSES

THURSDAY **3:00 to 4:30 P.M.** **October 3, 1957**

Diagnostic Roentgenology		Nuclear Medicine, Therapeutic Radiology, Miscellaneous	
300—Caffey	Normal Variants in the Growing Skeleton of Diagnostic Significance	**320—Fletcher and Bloedorn**	Radiotherapeutic Management of Oral Cavity and Oral Pharynx Cancers
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CONDENSED SCHEDULE OF COURSES

FRIDAY **1:30 to 3:00 P.M.** **October 4, 1957**

Diagnostic Roentgenology		Nuclear Medicine, Therapeutic Radiology, Miscellaneous	
400—Hodes	Recent Advances in Body Section Radiography	**420—Garcia**	The Treatment of Carcinoma of the Cervix
401—Dennis	Problems in Urinary Tract Diagnosis		
402—Felson	Some Fundamentals in Chest Roentgen Interpretation	**421—Wittenborg**	Radiotherapeutic Aspects of Tumors in Infants and Children
403—Ruzicka	Portal Venography—Techniques, Interpretation and Clinical Application		
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405—Margulis	Arteriography of Extremities		
406—Miller	Roentgen Examination of the Stomach and Small Bowel	**423—Evans**	Radiation Biology
407—Van Epps	Diagnosis of Pneumonias in Infancy and Early Childhood		
408—Greitz	Pneumography in Expanding Lesions of the Brain		

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Circulatory Changes During Muscular Work in Man. The Scandinavian Journal of Clinical & Laboratory Investigation, Supplement 24. By Alf Holmgren. Paper. Pp. 97, with 26 illustrations. The Scandinavian Journal of Clinical & Laboratory Investigation, Medical Department A, Rikshospitalet, Oslo, Norway, 1956.

During the transition from rest to work there is an initial fluctuation (increase-decrease-increase) in the brachial arterial pressure.

During muscular work with increasing load there is an approximately linear relationship between the arterial blood pressure and the load. The systolic pressure (measured after six minutes of work) increases an average 8 mm. Hg per 300 kg./min. There is less change in the mean and diastolic pressure. During thirty minutes of heavy work the systolic blood pressure decreases progressively about 6 mm. Hg after the initial minute of work. The difference between central and peripheral systolic pressure increases during work.

The peripheral venous pressures varied independently of central venous pressure in 4 of 5 cases during work. Muscular work with increasing load produced no significant change in central venous pressure. It appears to the investigator that no significant pooling occurs on the venous side and the filling pressure remains unchanged and is sufficient, although the duration of diastole decreases with increasing pulse frequency during work. This is in agreement with previous workers that right auricular and pulmonary wedge pressures are insignificantly affected by exercise in normal subjects. Therefore we assume that abnormal elevation of these pressures is associated with pathologic myocardial and/or valve function.

Jan Nyboer, M.D.

Diseases of the Heart. By Charles K. Friedberg, M.D., Attending Physician, The Mount Sinai Hospital, New York; Associate Clinical Professor of Medicine, College of Physicians and Surgeons, Columbia University. Second edition. Cloth. Price, $18.00. Pp. 1,161, with 157 illustrations. W. B. Saunders Company, 218 West Washington Square, Philadelphia 5, Pennsylvania, 1956.

In this second edition the author has revised the entire text, has included comprehensive discussions of the newer developments in our knowledge of heart disease, and has added three chapters dealing with graphic methods of examination.

Cardiac surgery in its many aspects, as well as the numerous advances in medical treatment, are fully and ably presented. Controversial questions such as those concerning cholesterol control for atherosclerosis, the use of anticoagulants, chair versus bedrest and the influence of hypotensive agents, are accorded fair, objective consideration that should be helpful to the reader.

In all, this is a most practical presentation on heart disease that should make an excellent reference work for the practicing physician.

William S. Reveno, M.D.

Über die Technik der simultanen Telefilmplanigraphie. Acta radiologica, Supplement 137. By Vidar Backlund. Paper. Price, Sw. Kr. 25:—. Pp. 124, with 39 illustrations and 13 tables. Acta radiologica, Stockholm 2, Sweden, 1956.

Conventional single-layer laminagraphy, when used to investigate multiple planes, is a time-consuming procedure, and delivers a relatively high dose of radiation to the patient. The theoretical formulation of Ziedses des Plantes in 1932, and the practical application of Manoel de Abreu in 1947, have initiated the so-called "simultaneous" procedures, in which several films, superimposed and adequately spaced within a special cassette, are all exposed during a single cycle of motion. The development of a workable device of this type is described in this monograph.

By increasing the target-skin distance ("tele"-laminagraphy), scattered radiation from the examined object can be reduced to acceptable limits, provided the surface under consideration is of 50–150 cm.^{2} For larger areas it becomes necessary to add a reciprocating grid. The intensifying screens must be very radiolucent,

and increasingly luminescent, the further away from the tube they are to be placed. Double emulsion films were used, but only single screens of calcium wolframate were placed on the side of the emulsion located away from the tube. Tinfoil applied to the back of each screen served to absorb the characteristic K-radiation emitted by tungsten. The intervals between the layers, which must maintain a certain relationship with the target-skin distance, are determined by the organ under consideration. As a rule, the smaller the object, the thinner the sections. Spacing was achieved with "leaves" of balsa wood and special cassettes for each type of layering. The installation used was a Siemens Universal Planigraph, modified to allow changes in target-skin distance, to permit the adaptation of a bulky cassette holder, and to include a device for magnification techniques. The author recommends using an automatic film-processing machine to assure uniform blackening of the "layers" and to step up the output so that 5 to 10 patients may be examined in one hour. He has used "simultaneous" laminagraphy on about twenty organs or parts of the body. He speaks of over 15,000 examinations in more than 10,000 patients, completed since he started using this method. A clinical evaluation of the procedure will be published at a later date.

The reproductions included appear comparable in quality to what can be obtained with the conventional uni-layer method. The monograph will prove to be of distinct value to all those who contemplate using any of the "simultaneous laminagraphy" devices.

E. R. N. Grigg, M.D.

Observations on the Migration of Some Labelled Substances Between the Urinary Bladder and the Blood in the Rabbit. Acta radiologica, Supplement 135. By Sven Erik Englund. Paper. Price, Sw. Kr. 25:—. Pp. 80, with 50 illustrations. Acta radiologica, Stockholm 2, Sweden, 1956.

This volume describes an experimental study to "estimate the degree of penetration—of various radioactive substances from bladder to blood and vice versa," using the rabbit as the experimental animal. For the study the radioisotopes Na^{24}, P^{32}, K^{42} and Br^{82} were chosen.

Following a review of the literature and a discussion of radioisotopes and detection techniques, the author outlines the method of carrying out the experimental problems under controlled conditions. There is a section on the anatomy of the rabbit bladder, with the surgical procedure used for instilling the material in question into the bladder or blood, and the method used for recovery of the isotope from the blood or bladder.

The first series of experiments entailed the absorption of P^{32}, K^{42}, Br^{82} and Na^{24} from the bladder singly, doubly mixed, and under varying controlled conditions. Quantitative attempts at measurement were made. A second series of experiments was designed to show the migration of radioisotopes from blood to bladder. The third series measured the differences in clearance of Br^{82} from an intramural and an intravesical site. The degree of migration of Br^{82} from an experimentally damaged bladder to blood was also evaluated.

In conclusion, the author felt his experiments demonstrated without doubt that "definite though small amounts of Na^{24}, P^{32}, K^{42} and Br^{82} penetrate the bladder mucosa in both directions." Experiments with the intramural and intravesical clearances of Br^{82} would appear to show "a barrier somewhere in the bladder mucosa" and to demonstrate that "this barrier lies in the vicinity of or in the bladder epithelium." The rate of absorption appears dependent on vascular mechanisms. Damage of the bladder mucosa using turpentine resulted in increased bladder-to-blood migration.

This material should be of interest to both radiologists and urologists, as it relates to the recent clinical instillations of radioactive materials in bladder neoplasms and other conditions. Again, attention may be directed to the difference of absorption from the bladder between one intact and one with an injured mucosa. Others concerned with research on radioactive substances will find in this monograph an excellent discussion of radioisotopes, techniques, and errors.

Robert C. Thumann, M.D.

Biophysical Investigations of Urinary Calculi; An X-Ray Crystallographic and Microradiographic Study. Acta radiologica, Supplement 133. By Curt Lagergren. Price, Sw. Kr. 25:-. Pp. 71, with 33 illustrations. Acta radiologica, Stockholm 2, Sweden, 1956.

This monograph describes the result of an investigation during which 600 urinary tract calculi were subjected to roentgen crystallography, microradiography and roentgen microdiffraction. These stones were divided into groups as to the site of origin in the kidney, ureter, or bladder.

The three methods of study, particularly roentgen microdiffraction, permit more satisfactory identification of the various chemicals involved than was possible with the usual chemical investigation. In addition, the present study shows the orientation of the specific chemical compounds within the various layers in any given calculus. For example, differentiation of three types of calcium phosphate which could not be separated chemically was possible, and in addition, each could be identified in a very thin layer in a laminated calculus. Eleven different compounds were found to constitute the usually occurring calculi. An important observation was the fact that any one compound, when it was deposited in a stone, was in a layer, or some other form, discretely separated from the other compounds. This is considered an expression of the changes in character of the urine which occur during formation of a given calculus. Further correlation of this with other urinary studies may indicate ways of preventing formation of a certain type of stone in a given individual.

This monograph is well documented. There are numerous illustrations of the different types of stone, the materials found in the various layers, and various types of roentgen diffraction patterns. The study represents an important addition to investigation of the urinary tract and will be of considerable interest to urologists and physiologists, in particular.

E. F. Lang, M.D.

BOOKS RECEIVED

Souvenir Number of The Indian Journal of Radiology, 1956; In Commemoration of the Diamond Jubilee of the Discovery of X-Rays by Professor Dr. Wilhelm Conrad Röntgen. K. P. Mody, Editor-in-Chief. Paper. Pp. 702, with numerous illustrations. Indian Journal of Radiology, Navsari Building. Dr. Dadashoy, Naoroji Road, Fort, Bombay 1, India, 1956.

International Congress of Gastroenterology. Fifth Meeting of L'Association des Sociétés Européenes et Méditerranéennes de Gastro-Entérologie. London, July 13–21, 1956. Cloth. Pp. 634, with numerous figures and tables. S. Karger, 215 Fourth Avenue, New York 3, New York, 1957.

Textbook of Pathology; With Clinical Applications. By Stanley L. Robbins, M.D., Associate Professor of Pathology, Boston University School of Medicine; Associate Director of the Mallory Institute of Pathology, Boston, Massachusetts; Lecturer, Harvard Medical School and Tufts University School of Medicine. Cloth. Price, $18.00. Pp. 1,351, with 933 illustrations. W. B. Saunders Company, 218 West Washington Square, Philadelphia 5, Pennsylvania, 1957.

Selected Papers from the Institute of Cancer Research: Royal Cancer Hospital and from the Royal Marsden Hospital, 1954. Published by order of the Committee of Management of the Institute and the Board of Governors of the Royal Marsden Hospital, London. Volume 9. Cloth. Pp. 904, with numerous illustrations. Chester Beatty Research Institute, Fulham Road, London, S.W.3, England, 1957.

Annual Review of Medicine. Volume 8. 1957. David A. Rytand, Editor. Cloth. Price, $7.00. Pp. 530, with illustrations and tables. Annual Reviews, Inc., Stanford, California, 1957.

Radiation: What It Is and How It Affects You. By Jack Schubert, Ph.D., and Ralph E. Lapp, Ph.D. Cloth. Price, $3.95. Pp. 314. The Viking Press, Inc., 625 Madison Avenue, New York 22, New York, 1957.

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Head

Neiss, A. Nachweis der Foveola pharyngea occipitalis im Röntgenbild und ihre Beziehung zum Canalis basilaris medianus. (Roentgen visualization of foveola pharyngea occipitalis and its relation to canalis basilaris medianus.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, March, 1957, *86*, 343–345. (Address: Anatomisches Institut, Krankenhausstr. 9, Erlangen, Germany.)

The pharyngeal foveola, a small, round, well defined defect in the base of the occipital bone was noted in 410 of routine sinus examinations. It was usually seen in adolescent females and more commonly in Negroes and the yellow race than in white patients. The canalis basilaris medianus which is in intimate relationship with the notochord is immediately adjacent to the pharyngeal foveola.

The generally held opinion that the foveola is formed by the exit of the notochord is doubted because it is never seen in newborns, but appears only in adolescence.—*Frederick M. Reis, M.D.*

Neck and Chest

Motta, G., and Piazzi, M. L'indagine radiologica nei tumori della loggia io-tiro-epiglottica. (The radiologic image of tumors of the hyo-thyro-epiglottic space.) *Ann. radiol. diag.*, 1956, *29*, 432–481. (From: Clinica Otorinolaringologica and Istituti di Radiologia e del Radio dell'Università, Bologna, Italy.)

Since this particular part of the upper respiratory tract is difficult to examine carefully either by mirror or endoscopic examination, the authors have studied tumors and extension of tumors in this space by a combination of roentgenographic techniques. Usually no definite symptoms develop to lead the examiner to suspect that the laryngeal vestibule is involved.

A series of 9 cases with involvement of the hyo-thyro-epiglottic space was studied by the method proposed by the authors and the roentgen findings were compared with the surgical specimen in each case with fairly accurate correlation.

The routine lateral projection of the neck was used to measure the size of the hyo-thyro-epiglottic space and to demonstrate if any posterior displacement of the epiglottis has occurred. Also, if any differences in opacity of these structures or any irregularity of outline and finally if any ulcerations or fissures were present. Another lateral exposure was made with the patient executing a Valsalva maneuver, so as to better visualize the area posterior to the epiglottis. Serial laminagrams were taken after a swallow of a thin barium mixture to show ulcerations or fissures extending within the larynx.

Thus the authors believe that by following this method of examination, extension of laryngeal tumors into the hyo-thyro-epiglottic area can clearly be demonstrated, together with the depth of the extension. With this information surgical or radiotherapeutic procedures can be more carefully planned and executed.

The illustrations in this article are numerous and fairly good. It is the reviewer's impression that it would take considerable experience to learn proper interpretation of the roentgen findings in following such a procedure.—*Peter E. Russo, M.D.*

Powers, William E., McGee, Harry H., Jr., and Seaman, William B. Contrast examination of the larynx and pharynx. *Radiology*, Feb., 1957, *68*, 169–178. (Address: W. E. Powers, 510 S. Kingshighway, St. Louis 10, Mo.)

The authors report the results of a study of the normal and abnormal anatomy and physiology of the larynx and pharynx, and describe a technique of examination utilizing topical anesthesia and contrast medium. Anesthetization of the larynx is essentially that used in bronchography. Five to 10 cc. of opaque medium (lipiodol, dionosil in aqueous suspension or dionosil oily) are dripped slowly over the back of the tongue intermittently during inspiration. The medium fills the valleculae and pyriform sinuses and coats the larynx. Posteroanterior and lateral spot-films are exposed while the patient performs various maneuvers.

This technique was employed to study the anatomy and function of the hypopharynx and larynx in 25 normal subjects. Ten patients with unilateral laryngeal palsy and a number of others, with a variety of tumors involving the hypopharynx and larynx, were also studied.

Excellent roentgenograms, demonstrating the normal and the abnormal appearance, are reproduced.—*Arno W. Sommer, M.D.*

Scadding, J. G. Pulmonary fibrosis and collagen diseases of the lungs. Symposium. I. Clinical problems of diffuse pulmonary fibrosis. *Brit. J. Radiol.*, Dec., 1956, *29*, 633–641. (From: The Institute of Diseases of the Chest, Brompton, London, S.W.3, England.)

The term "collagen disease," as originally proposed, was not designed to be used as a diagnostic symbol relating to a complex of functional and structural disorders characteristic of morbid entities. It merely called attention to certain diseases with obscure etiology, distinguished by a systemic alteration of the connective tissue, specifically, of its intercellular components.

It is, therefore, not to be expected that any conformity of roentgenologic patterns in the lungs exists.

The 2 basic characteristics of the collagen diseases are: (a) The fundamental involvement of connective tissue in such a way that fibrosis may follow, and (b) the involvement of vascular structures.

Polyarteritis nodosa: A variety of roentgenologic appearances—diffuse mottling, areas of confluent consolidation, coarse striation radiating from enlarged hili, rounded opacities of low density (2 to 5 mm. in diameter) and occasionally a small costophrenic shadow denoting a pleural reaction or a large one indicating fluid; these emphasize the transient nature of the lung shadows.

Another roentgenologic feature of polyarteritis is one which may equally well occur in the eosinophilic infiltrations of asthma; namely, the formation of a pseudocavity or pneumatocele.

The "batswing" shadow described by Doniach (1947), Hodson (1950) and others is associated with azotemic edema. It constitutes a roentgenologic peculiarity which has never been satisfactorily explained. The unusual distribution of the opacities is an expression of the difference in vascular anatomy between the "medulla" of the lung and its "cortex" or "mantle."

In this condition, which most often affects women between forty and fifty, the typical lesion is the facial rash. One of the most constant features is polyserositis. This naturally influences the chest roentgenologic picture considerably. There is nothing specific about these lesions.

Scleroderma: Though manifesting itself mainly in the skin, it is now widely recognized as involving many systems, particularly the alimentary canal. This consists of an interstitial-like infiltrative process in the central portions of both lower lobes, changes which tend to clear and recur with increased distribution at each recurrence. Later they become widespread and permanent with progressive fibrosis. Lung changes are uncommon in scleroderma.

Rheumatic fever and rheumatoid disease: Rheumatic pneumonia is a rare finding. The authors classify the roentgenologic appearance into 2 groups: (1) Extensive consolidation in one or both lower lobes, more commonly the left, persisting for varying periods from one week up to several months and (2) a type of consolidation which is focal, multi-lobar, evanescent and migratory. In cases in which a severe infection exists, death may occur.

The prognosis in rheumatic fever is grave if extensive pulmonary manifestations are present.

In rheumatoid arthritis, pleural effusion is uncommon except when heart failure has supervened.—*Stephen N. Tager, M.D.*

Gough, J. Pulmonary fibrosis and collagen diseases of the lungs. Symposium. II. Generalised and primary fibrosis of the lungs. *Brit. J. Radiol.*, Dec., 1956, *29*, 641–645. (From: Department of Pathology, Welsh National School of Medicine, Cardiff, Wales.)

A variety of conditions which fall into 2 categories are discussed in this article. They are either forms of diffuse fibrosis confined to the lungs, or forms associated with systemic involvement of connective tissue, including certain so-called collagen diseases.

In some instances fibrosis is associated with dilatation of air spaces giving the appearance of honeycomb or cystic lung. In other instances the air spaces are obliterated in the affected areas giving a solid or compact fibrosis. The latter type occurs when there has been organization of much exudate in the air spaces.

Diffuse fibrosis of Hamman and Rich: This is a specific lung lesion consisting of a diffuse progressive interstitial proliferation of fibrous tissue with hyaline membranes lining the alveoli and with eosinophile leukocytes in the interstitial tissue in most cases. Cyanosis and cor pulmonale develop. The cause of Hamman-Rich fibrosis is not known. No bacteria or other organisms have been found in the lesions. The prognosis is hopeless. Death is due to cardiac or respiratory failure. At autopsy, the lesions are found to be extensive throughout both lungs.

Diffuse interstitital fibrosis due to granuloma: More common than the Hamman and Rich type of fibrosis is a chronic form associated with cystic (honeycomb) appearance. The onset is insidious. Respiratory or cardiac symptoms develop slowly over a period of years.

Scleroderma: The association of pulmonary fibrosis with scleroderma is now well recognized.

The lungs are small with depressions on the surface—pneumolobatum. Two types of change are found in the lung. One consists of diffuse fibrosis associated with honeycomb appearance—cystic pulmonary sclerosis. This is characteristically found in the bases of the lungs with prolongation up to the hilus. The other change consists of more solid or compact areas of fibrosis. The solid areas are all due to collapse and induration as judged by the elastic tissue stains.

Lupus erythematosus: The most common lesions are of a nonspecific type including bronchopneumonia, hemorrhage, pleural effusion and edema.

Disseminated lupus erythematosus does not present a characteristic appearance either on microscopic examination of pulmonary tissue or on roentgenographic examination of the chest.

Periarteritis nodosa: In the classical form of this condition the lungs were not involved.

Lung changes associated with rheumatoid arthritis: Specific changes in the form of rheumatoid nodules have seldom been found in the lungs.

In those instances where roentgenograms had shown little or no evidence of pre-existing pneumoconiosis, the characteristic findings in the rheumatoid cases were circumscribed opacities. When, however, the incidence of pneumoconiosis had been higher there was greater tendency for the development of what appeared in the roentgenograms to be ordinary massive fibrosis.

Clinical findings will not be adequate in all cases to explain a pattern of diffuse shadows in lung roentgenograms. Biopsy is therefore being employed with increasing frequency in such cases.—*Stephen N. Tager, M.D.*

Lodge, Thomas. Pulmonary fibrosis and collagen diseases of the lungs. Symposium. III. Pulmonary fibrosis and the collagen diseases: Radiological aspects. *Brit. J. Radiol.*, Dec., 1956, *29*, 645–656. (From: The Royal Hospital, Sheffield, Yorkshire, England.)

The authors list 7 possible causes of shadows, such as diffuse reticular or nodular opacities, in the chest roentgenogram.

The occupation history may be sufficient to provide a diagnosis beyond reasonable doubt. Similarly a history of residence in certain parts of the world may give the clue to certain diagnoses which otherwise would be unlikely: *e.g.*, histoplasmosis.

Important clues may be found in the skin. In sarcoidosis there may be obvious changes in the form of gross nodular sarcoid infiltrations on the limbs or trunk, or even more obvious changes, such as lupus pernio of the face. One variety of diffuse involvement of the lungs which eventually gives rise to a honeycomb appearance is associated with mesodermal dysplasia of the tuberous sclerosis-adenoma sebaceum group. Lupus erythematosus in its disseminated form is another skin condition associated with diffuse pulmonary infiltration. A curious diffuse fine mottling of the lung has been observed in 2 cases of pseudoxanthoma elasticum.

Evidence of underlying disease in other systems may be obtained on routine physical examination. The presence of mitral stenosis will give a clue to the diagnosis of hemosiderosis. In polyarteritis nodosa, lung changes, if they appear, are generally only part of a complex clinical picture.

The clinico-roentgenologic picture of sarcoidosis can be very characteristic. In the early stage, the typical sequence of enlarged hilar lymph nodes followed by the appearance of a diffuse mottling in the lungs as the lymph nodes subside, in a patient without serious symptoms and with slight or no cutaneous sensitivity to tuberculin, is sufficient to establish the diagnosis for practical purposes. In the late stage, the rather linear type of fibrosis in the middle zones, with emphysema and remnants of the original nodular infiltration at the apices and bases, are unlikely to be mimicked by any other condition. The enlarged hilar shadows and diffuse reticular shadows extending out from them, which are produced by lymphangitis carcinomatosa, may mimic the appearance of an intermediate stage of sacroidosis rather closely; but the symptoms will be quite different. The patients with lymphangitis carcinomatosa have steadily progressive dyspnea, which soon becomes severe with characteristically rapid and shallow breathing. An alimentary disorder which may give rise to diffuse pulmonary shadows is esophageal obstruction. In most instances, this will be symptomatically obvious. But in some cases of achalasia of the cardia, the patient may not complain of dysphagia even though his esophagus is grossly dilated and spilling over into his lungs at night, eventually giving rise to widespread fibrosis. Accordingly, in obscure cases of pulmonary fibrosis, it is a good practice to request an examination of the esophagus after barium swallow study.

The condition of chronic diffuse interstitital fibrosis of the lung associated with the names of Hamman and Rich, presents such a typical clinico-roentgenologic picture that it can be recognized with a high degree of certainty.

Routine laboratory investigations rarely give important aid in diagnosis in obscure cases. Examination of the blood is rarely helpful.

In many instances, the diagnosis will first be established by biopsy of some reasonably accessible tissue. It is worthwhile of making a careful search for enlarged lymph nodes in any site. In suspected polyarteritis nodosa, muscle biopsy is frequently performed. This procedure is disappointing unless there is some clinical evidence of abnormality in the muscles, such as pain, tenderness, palpable nodules or induration.

Chronic nodular fibrosis of the lungs: These all present a clinical picture of progressive dyspnea, cyanosis, clubbing of the fingers and diffuse reticulonodular shadows in the chest roentgenogram.

The more chronic type constitutes a clinically recognizable group. The cause remains unknown. The authors prefer a noncommittal descriptive term for the more chronic types, such as "chronic diffuse interstitital fibrosis."

Idiopathic pulmonary hemosiderosis was first described in children, in whom the clinical picture is one of periodic attacks of dyspnea and cyanosis, associated with hypochromic anemia with hemolytic features. Roentgenologically, there is a diffuse fine stippling in the lungs, which is more dense at the time of an attack, and diminishes thereafter. It may resolve completely between attacks. Pathologically, there is a fibrotic reaction around focal deposits of hemosiderin. The prognosis is bad.

Polyarteritis nodosa is an unsatisfactory diagnosis, since it states no more than that certain histologic changes are found in the smaller arteries and arterioles. It conveys no information about etiology. Several sorts of involvement of the lung exist in polyarteritis nodosa. One consists of diffuse changes giving rise to a fine nodular pattern. Another type of lung change consists of the effects of changes in the larger vessels, giving rise to infarcts, and producing shadows of considerable size. It should be remembered that of all the causes of chronic diffuse pulmonary fibrosis only one—tuberculosis, is curable. Some of the conditions found may be improved by the administration of cortisone or ACTH, *e.g.*, disseminated lupus erythematosus, polyarteritis nodosa

or lung changes associated with rheumatoid arthritis.—*Stephen N. Tager, M.D.*

Feinberg, S. B., and Goldberg, M. E. Hyaline membrane disease: Preclinical roentgen diagnosis; a planned study. *Radiology*, Feb., 1957, *68*, 185–192. (Address: S. B. Feinberg, University of Minnesota Medical School, Minneapolis 14, Minn.)

Infants with hyaline membrane disease are usually premature, delivered by cesarean section, or born of a diabetic mother. In a matter of minutes to hours after birth, breathing becomes labored, as if against obstruction, and death may ensue. Grossly, the lungs are markedly engorged and contain few or no gross air-filled spaces. Microscopically, the capillaries are engorged, and the alveoli and alveolar ducts may be collapsed. The few remaining air spaces are lined with a hyaline-like material.

A study was undertaken to determine whether the diagnosis of hyaline membrane disease could be made before the development of clinical symptoms, thereby facilitating prompt treatment. Roentgenograms of all premature infants, those born of diabetic mothers, and those delivered by cesarean section were made within the first hour of life and follow-up roentgenograms were taken at 2, 4, 8, and 24 hour intervals. Of 89 infants followed, 8 had histologically proved hyaline membrane disease and 2 had apparent disease and survived. In 7 of the 8 proved cases, unequivocal abnormality was demonstrable on the initial roentgenogram and the other case showed signs suggestive but not diagnostic of the disease. In 2 infants early roentgen and later clinical signs of the syndrome eventually cleared. The early roentgenograms revealed a finely granular pattern symmetrically distributed throughout both lung fields and abnormal prominence of the linear hilar and perihilar bronchovascular strands. Follow-up examinations showed gradual progression to the obvious granularity described in the literature with eventual development of frank localized or generalized atelectasis. In 7 of the 10 cases, symptoms developed from fifteen minutes to three hours after birth. The lag between roentgen findings and clinical manifestations was nine hours in one case and six hours in another.—*Harold C. Hamilton, M.D.*

di Rienzo, S., and Pereira Duarte, R. O. Die Bronchographie mit Barium. (Bronchography with barium.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, March, 1957, *86*, 315–318. (Address: S. di Rienzo, Avenida General Paz 151, Cordoba, Argentina.)

Barium-carboxymethylcellulose surpension used in bronchography permits as good visualization as that achieved with iodized oils or carboxymethylcellulose-iodine preparations, but has the advantage of being less toxic, nonabsorbable and very inexpensive. There is less irritation of the bronchial tree, good "foliage effect" and no fogginess because this preparation is not absorbed. The viscous barium is immediately expelled by coughing and no trace can be found after forty-eight to seventy-two hours. No toxic or allergic reactions have been encountered. Re-examination of the chest is not hampered by residual densities from previous bronchography. The extremely low cost should be of particular advantage to large chest clinics.—*Frederick M. Reis, M.D.*

Cohen, Samuel, Perlberg, Harry J., and Larde-Arthez, C. R. The use of visciodol in bronchography. *Radiology*, Feb., 1957, *68*, 197–203. (Address: S. Cohen, B. S. Pollak Hospital for Chest Diseases, Jersey City, N. J.)

The authors report the results of their experience with a mixture of lipiodol and finely divided sulfanilamide, called visciodol, used for bronchography in 82 cases. The material was found to be nonirritating and rapidly eliminated from the bronchi. Successful repeat bronchograms could usually be obtained within a week after initial instillation, with no halo effect from the residual contrast medium. Due to the increased viscosity of this mixture, tissue surfaces were well coated, but alveolar filling usaully did not occur. The bronchial outlines could therefore be visualized with greater clarity. No untoward effects were observed.—*Arno W. Sommer, M.D.*

Pontius, John R., and Jacobs, Lewis G. The reversal of advanced bronchiectasis. *Radiology*, Feb., 1957, *68*, 204–208. (Address: J. R. Pontius, VA Hospital, 13th and Harrison Sts., Oakland 12, Calif.)

Reversible bronchiectasis has been reported by a number of authors. The cases have, however, been limited largely to the acute and subacute phases of the disease. Also, all have been of the cylindrical type.

The authors describe a case in which abnormal pulmonary densities were observed in 1953 and abnormal bronchograms, showing bronchiectasis of cylindrical and saccular type, were obtained in 1954 and 1955. Severe chronic bronchographic abnormalities existed for at least ten months under observation. Complete reversal of these changes followed two months of intensive medical measures. Whether or not this reversal was related to the management or to some external cause could not be determined. Also, the permanence of the clearing is open to question.

As regards the possibility that the last set of roentgenograms might have been of a different individual examined through error, the authors point out that careful study of the roentgenograms taken on all examinations convinced them that the same patient

was examined on all occasions and also, that the same bronchi were seen on the various examinations. This would seem to prove that the saccular bronchiectasis did disappear, and the bronchi reverted to normal.—*A. Ralph Watson, M.D.*

Bluth, Irwin. A note on the roentgen features of bronchial adenoma of the peripheral type. *Radiology*, Feb., 1957, *68*, 193–196. (Address: Beth-El Hospital, Rockaway Parkway at Avenue A, Brooklyn 12, N. Y.)

Since bronchial adenomas are thought to arise in the mucous glands, they occur more frequently in the main bronchi in which such glands are abundant. They may occasionally, however, occur more peripherally in the lesser bronchi. Although generally considered benign, they may on occasion present malignant features and invade or metastasize to the bronchial or tracheobronchial lymph nodes or be found in isolated lymphatics in the mediastinum. Spread to the liver, kidneys, and vertebrae has been reported. Bronchoscopy is of great value in diagnosing the centrally located adenoma, but is of no value when the tumor is peripherally situated.

Roentgenographically, the peripheral tumor usually appears as a circumscribed density with a suggestion of lobulated borders. Size varies from 1.5 to 8.0 cm. in diameter. The differential diagnosis must include a peripherally located primary carcinoma of the lung, a solitary metastasis, and other benign tumors such as hamartoma.

The author reports 3 cases of the peripheral type of bronchial adenoma. In each case the tumor was an incidental finding on the chest roentgenogram and there were no clinical symptoms referable to the lesions. In none of these was the diagnosis established until the resected specimen had been analyzed by the pathologist. It is mentioned that calcification in a nodule should not be a basis for differentiation among the various benign growths since tuberculoma and hamartoma are sometimes calcified. Cited is an authentic case, as yet unreported, of a peripheral adenoma with calcification.—*Donald N. Dysart, M.D.*

Salinger, Hans, and Friedman-Barou, Hannah. The use of different tube shifts in lung tomography. *Radiology*, Feb., 1957, *68*, 209–216. (Address: H. Salinger, 3 Bialikstr., Tel Aviv, Israel.)

The factors determining whether blurred shadows of objects situated at a short distance from the layer of interest in laminagraphy remain or are eliminated more or less completely are: (1) The distance between the object casting the shadow and the section plane. Increased distance results in more complete blurring; (2) the roentgen-ray absorbing power of the object. The denser the shadow the more difficult it is to eliminate; (3) the length of the tube-film shift. The greater the movement, the better the blurring; and (4) the direction of the long axis of the object the shadow of which is to be eliminated. When this axis is parallel to the tube-shift, elimination of its shadow is more difficult and often impossible.

The fourth factor is the one most easily influenced. In laminagraphy performed with rectilinear shift, the incompletely blurred shadows of structures with a long axis in the same direction as the shift may obscure important findings and prevent their recognition. By the addition of an oblique tube-film shift to the routine craniocaudal shift in lung laminagraphy, valuable additional information may often be obtained. With this method it is important that the incidence of the central ray and the respiratory expansion of the lung be the same in all laminagrams. The apex of the lung and the postoperative narrowed chest are well adapted to examination by this method.

Three cases illustrating the advantages of this method are presented.—*Harold C. Hamilton, M D.*

Scheff, Saul, Bednarz, Wallace W., and Levene, George. Roentgenologic aspects of retropleural hematomas following sympathectomy. *Radiology*, Feb., 1957, *68*, 224–230. (Address: S. Scheff, Massachusetts Memorial Hospitals, 750 Harrison Ave., Boston 18, Mass.)

The roentgenologic appearance and incidence of retropleural hematomas, following operations on the sympathetic nerve trunks in 590 operations, are described. Postoperative roentgenograms were obtained on 220 of the 300 patients included in this series. Retropleural hematomas were found in 40 (18 per cent).

Retropleural hematomas are relatively innocuous. They are important chiefly because they may be confused with more serious intrathoracic lesions. Roentgenologically the hematoma appears as a smooth-bordered or slightly lobulated mass arising from the posterior thoracic wall. On a posteroanterior roentgenogram the medial border of this mass is seen to blend with the mediastinal shadow. As resorption of the hematoma occurs, the previously smooth border becomes irregular and ill-defined. In most instances roentgenologic evidence of the hematoma will disappear within four months, but there may be some residual fibrosis and pleural thickening.—*Arno W. Sommer, M.D.*

Fossati, Franco, and Papagni, Leonardo. Sulla deviazione "a falce" del terzo inferiore dell'esofago. ("Sickle" shaped deviation of the lower third of the esophagus.) *Radiol. med.*, Feb., 1957, *18*, 113–122. (From: Istituto di Clinica Medica Generale dell'Università, Milano, Italy.)

The authors report a series of 4 cases in which the lower third of the esophagus was deviated anteriorly and to the left giving a sickle-shaped appearance. In each of these cases the cause for the deviation was elongation with arteriosclerotic changes of the thoracic aortic. In only one of the 4 cases was dysphagia present.

This deviation of the esophagus should not be mistaken for either intrinsic or extrinsic lesions of the esophagus, or for mediastinal tumors which may arise in the posterior mediastinum. Routine posteroanterior and lateral roentgenograms with the esophagus filled with barium are most helpful in making a diagnosis. Aneurysm of the aorta at this level usually produces a more profound deviation of the esophagus or displaces the esophagus to the right of midline.

This article contains 6 excellent anatomic diagrams to show the relationship of the aorta and esophagus at this level—and also how the "sickle" deviation occurs. The reproduction of roentgenograms of their cases is of excellent quality.—*Peter E. Russo, M.D.*

de Almeida Prado, A. (São Paulo, Brazil.) "Maladie de Chagas" et "mal de engasgo" (qui empêche d'avaler) dans leurs rapports avec l'achalasie. (Chagas' disease and dysphagia in their relation with achalasia.) *Presse méd.*, March 20, 1957, *65*, 521–523.

In Chagas' disease the causative organism, Trypanosoma cruzi, causes myocarditis with disorders of the conduction system of the heart. Mega-esophagus due to achalasia is said to be the result of involvement of the intramuscular plexus of Auerbach. In some patients with mega-esophagus, cardiac enlargement is also present. Granulomas and pseudocysts produced by T. cruzi have been demonstrated in the circular muscular layer of the lower esophagus, associated with cicatricial changes in Auerbach's plexus.

This association is noted in Brazil and apparently is quite rare except in those areas where trypanosomiasis is endemic.—*Charles M. Nice, Jr., M.D.*

Gynecology and Obstetrics

Kräubig, H. Die röntgenologische Darstellung des Kindes in utero durch die Fetographie. (Roentgen visualization of fetus in utero by fetography.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, March, 1957, *86*, 351–356. (From: Universitäts-Frauenklinik, Göttingen, Germany.)

Visualization of fetal soft tissues is achieved by injection of 15–20 cc. of Immetal (Bayer) into the amniotic cavity. A 1.5 mm. long lumbar puncture needle is introduced slightly to the right or left and distal to the navel under strict asepsis. The patient is instructed to change her position frequently to distribute the contrast material. After six to seven hours, a posteroanterior or anteroposterior exposure is made. A fine film of contrast material is seen over the entire skin area of the fetus.

Indications for fetography are: (1) Hydramnion: (demonstration of fetal malformation as indications for cesarean section); (2) previous fetal malformations (especially when cesarean section is to be considered); (3) diabetic women; (4) erythroblastosis; and (5) question of fetal death (only conditional indication).

A history of erythroblastosis makes fetography necessary to rule out fetal hydrops which would make cesarean section necessary. Fetal hydrops is easily demonstrated by this method.

Possible complications are discussed and 4 cases illustrated. The author has had no reactions.—*Frederick M. Reis, M.D.*

Kaiser, Irwin H., and Marvin, James F. Effect of antepartum diagnostic roentgenography on the white blood cell count of the newborn infant. *Radiology*, Feb., 1957, *68*, 249–250. (Address: I. H. Kaiser, Department of Obstetrics and Gynecology, University of Minnesota Medical School, Minneapolis 14, Minn.)

The effect of antepartum diagnostic roentgenography, late in pregnancy, on the white blood cell count of the newborn infant was studied at the University of Minnesota Hospitals. Included in the study were 101 infants whose mothers had had no diagnostic roentgenography and 22 infants whose mothers had had roentgen-ray examinations late in pregnancy. Fourteen of the 22 infants were exposed to irradiation in the last fourteen days of pregnancy and 20 of the 22 exposures were within thirty-one days of delivery.

The results of the study indicate that diagnostic maternal roentgenography in late pregnancy does not produce significant changes in the white blood cell count of the newborn infant. The estimated total-body dose to the fetus from parallax pelvimetry was 4.4 kg. roentgens (assuming an average fetal weight of 3 kg.).—*Harold C. Hamilton, M.D.*

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